Annual Report Concerning Foodborne Disease in New Zealand, 2009

ANNUAL REPORT CONCERNING FOODBORNE DISEASE IN NEW ZEALAND 2009

Prepared for New Zealand Food Safety Authority under project MRP/09/04 – Systematic reporting of epidemiology of potentially foodborne disease in New Zealand for year 2009, as part of overall contract for scientific services

by

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ANNUAL REPORT CONCERNING FOODBORNE DISEASE IN NEW ZEALAND 2009

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1 INTRODUCTION

The New Zealand Food Safety Authority (NZFSA) has an aim to reduce food-related risks to human health. Its Science Strategy has identified human health surveillance as an essential element of the monitoring and review component of its risk management framework. In addition, evidence from notifications, case enquiries, outbreak investigations and other epidemiological studies of human enteric diseases are being increasingly used as sources of data for risk assessments. There is increasing interest in foodborne disease statistics within NZFSA and its stakeholders.

This report for the calendar year 2009 is intended to be part of a series providing a consistent source of data and method of presentation to allow monitoring of foodborne illness in New Zealand.

1.1 Human Health Surveillance Data and Foodborne Disease

The information in this report concerns reported cases of notifiable disease and reported outbreaks collected in the EpiSurv database (for a description of EpiSurv, see section 2.1.1 of this report). There are a number of notifiable illnesses which may be caused by transmission of pathogens in foods, but it is important to remember that most of the information concerns the illness, not the mode of transmission. The information needs to be considered with two caveats:

1. Notified cases of illness and reported outbreaks represent a subset of all the cases and outbreaks that occur in New Zealand each year. Many cases do not visit a GP or otherwise come to the attention of the medical system. By using these data as indicators, we are assuming that they are representative of all the cases and outbreaks that occur (see section 3 for a further discussion of this issue).
2. Foodborne transmission is only one of the routes by which humans are exposed to pathogens; other routes include water, animal contact and person to person. There are a number of indicators from which we can get information on the proportion of cases caused by foodborne transmission:

    - Reported risk factors: for a proportion of the notified cases, supplemental information is obtained by Public Health Units (PHUs) on risk factors. This information should be interpreted with some caution as it is self reported by cases, no external validation of this information is undertaken, and often the cases will report several potentially important risk factors. The quality of information from notifiable disease surveillance as an indication for foodborne disease transmission has been reviewed in more detail (Lake *et al.*, 2005).
    - Outbreak reports: the circumstances of an outbreak (multiple cases from a single event) means that investigation is more likely to identify a source of exposure to the pathogen. However, only a small proportion of outbreaks are reported, and experience shows that outbreaks associated with a foodservice premises are more likely to be reported and investigated.
    - Expert opinion: based on their experience in laboratories and epidemiological investigations, as well as knowledge of factors influencing the risk, experts can provide estimates of the proportion of cases caused by foodborne transmission. Estimates for New Zealand have been developed for some foodborne diseases (Cressey and Lake, 2005), as presented in relevant report sections. These are not fixed values; changes to the New Zealand food chain may require the values to be amended.

- Overseas analyses and estimates: information for countries with similar food supplies to New Zealand can be helpful, especially for illnesses where a foodborne estimate was not developed. Three sets of published expert opinion estimates are given in Table 1, for the USA (Mead *et al.*, 1999), Australia (Hall and Kirk, 2005) and the Netherlands (Havelaar *et al.*, 2008). It is worth noting that, although for most of the diseases included in this report foodborne transmission is considered significant, there are several illnesses (shigellosis, giardiasis, cryptosporidiosis, infection with Hepatitis A) where it is considered to be only a small proportion of the total.

Table 1: Overseas estimates of the food attributable proportion of selected microbial diseases

Illness/hazard	% Foodborne		
	USA	**Australia**	**Netherlands***
Bacteria			
Bacillus cereus	100	100	90
Campylobacter spp.	80	75	42
Clostridium perfringens	100	100	91
Escherichia coli O157:H7	85	65	40
Listeria monocytogenes	99	NE	69
Salmonella non-typhoidal	95	87	55
Shigella spp.	20	10	NE
Staphylococcus food poisoning	100	100	87
Yersinia enterocolitica	90	75	NE
Parasitic			
Cryptosporidium parvum	10	10	12
Giardia lamblia	10	5	13
Viral			
Hepatitis A	5	NE	11

* the Dutch study also collected opinions on the proportion of disease due to travel. A proportion of this will also be foodborne
NE = not estimated

This report considers information for the 2009 calendar year. Information from the scientific literature and other sources concerning food safety for that year has been summarised. However, the time taken to publish scientific information is often lengthy, and it may be that additional information becomes available in the future.

1.2 Conditions Included in Report

The conditions that have been selected for inclusion in the report are those that have:

1. The potential to be caused by foodborne transmission; and,
2. Available historical and current national data sources.

The potentially foodborne conditions that were selected for inclusion in this report are listed in Table 2. Data have been drawn from a number of sources including disease notification, hospitalisation, outbreak report and laboratory surveillance databases.

Table 2: Potentially foodborne conditions included in the report

Disease	Type	Source(s)	ICD*-10 code
Bacillus cereus intoxication	Bacterium	N, O, H	A05.4 Foodborne *Bacillus cereus* intoxication
Campylobacteriosis	Bacterium	N, O, H	A04.5 *Campylobacter* enteritis
Ciguatera poisoning	Toxin	N, O, H	T61.0 Toxic effect: Ciguatera fish poisoning
Clostridium perfringens intoxication	Bacterium	N, O, H	A05.2 Foodborne *Clostridium perfringens* [*Clostridium welchii*] intoxication
Cryptosporidiosis	Protozoan	N, O, H	A07.2 Cryptosporidiosis
Giardiasis	Protozoan	N, O, H	A07.1 Giardiasis [lambliasis]
Hepatitis A virus infection	Virus	N, O, H	B15 Acute hepatitis A
Listeriosis (total and perinatal)	Bacterium	N, O, H	A32 Listeriosis
Norovirus infection	Virus	O, H	A08.1 Acute gastroenteropathy due to Norwalk agent
Salmonellosis	Bacterium	N, O, H, L	A02.0 *Salmonella* enteritis
Scombrotoxicosis	Toxin	N, O	T61.1 Toxic effect: Scombroid fish poisoning
Shigellosis	Bacterium	N, O, H, L	A03 Shigellosis
Staphylococcus aureus intoxication	Bacterium	N, O	A05.0 Foodborne staphylococcal intoxication
VTEC/STEC infection	Bacterium	N, O, L	A04.3 Enterohaemorrhagic *Escherichia coli* infection
Toxic shellfish poisoning	Toxin	N, O	T61.2 Other fish and shellfish poisoning
Yersiniosis	Bacterium	N, O, H	A04.6 Enteritis due to *Yersinia enterocolitica*

Data Sources: EpiSurv notifications (N), EpiSurv outbreaks (O), MOH hospitalisations (H), ESR laboratory data (L)
VTEC = Verotoxin-producing *Escherichia coli* STEC = Shiga Toxin-producing *Escherichia coli*
* International Classification of Diseases

The notifiable conditions were selected for inclusion in the report where it was considered that a significant proportion would be expected to be foodborne or the disease organism has been

reported as the cause of foodborne outbreaks. *Salmonella* Typhi and *Salmonella* Paratyphi are not included as the majority of cases acquire their infection overseas.

For some diseases (intoxications from the bacteria *Bacillus*, *Clostridium* and *Staphylococcus*, and norovirus infection) not every case is notifiable; only those that are part of a common source outbreak.

For some conditions (campylobacteriosis, listeriosis, salmonellosis, VTEC/STEC infection, yersiniosis) the attribution of disease incidence to foodborne transmission was estimated by an expert consultation held on 24 May 2005 (Cressey and Lake, 2005). In the current report the proportions of food-associated cases, derived from expert consultation, have been used to estimate the number of food-associated cases of relevant diseases. In this process it has been assumed that travel-associated cases can be removed from the total cases before application of the food-associated proportion.

This report includes both notifiable diseases in the form of acute gastrointestinal illness, and sequelae which are considered to result from these preceding infections (Table 3). The two sequelae included in the report, haemolytic uraemic syndrome (HUS) and Guillain-Barré Syndrome (GBS) are severe illnesses and occasionally life threatening,

Table 3: Sequelae to potentially foodborne conditions included in the report

Disease	Source(s)	Comment
Guillain-Barré Syndrome (GBS)	H (G61.0 Guillain-Barré syndrome)	Sequelae following infection with *Campylobacter* [1]
Haemolytic uraemic syndrome (HUS)	H (D59.3 Haemolytic-uraemic syndrome)	Sequelae to infection with VTEC/STEC

Data Sources: MOH hospitalisations (H)

[1] While there is evidence that GBS can be triggered by other microbial infections (e.g. cytomegalovirus, Epstein-Barr virus, *Mycoplasma pneumonia*), *Campylobacter* infection is the only recognised triggering organism that is potentially foodborne

The data sources above have been selected on the basis of availability of data for the specified reporting period and their availability within the timeframe required for the report.

Some data such as official cause of death are not published until several years after the end of the year in which the event occurred (although deaths may be reported as part of the case notification data recorded in EpiSurv). For this reason these data cannot be included in a report published soon after the end of the calendar year.

2 METHODS

This section includes descriptions of the data sources, analytical methods used and comments on quality of data (including known limitations).

The report uses the calendar year (1 January to 31 December 2009) for the reporting period.

2.1 Data Sources

The key sources of data used in this report are detailed in the following sections.

2.1.1 EpiSurv - the New Zealand notifiable disease surveillance system

Under the Health Act 1956 health professionals are required to inform their local Medical Officer of Health of any notifiable disease that they suspect or diagnose. The current reporting year was the second year in which laboratories were also required to report notifiable disease cases to Medical Officers of Health. It is uncertain whether this change would have impacted on the numbers of notified cases, although data on salmonellosis (section 4.13.3.1) and shigellosis (section 4.14.3.1) suggest an increasingly good alignment between notified and laboratory confirmed cases in recent years.

Notification data are recorded using a web based application (EpiSurv) available to staff at each of the 20 public health units (PHUs) in New Zealand. These data are transferred to the Institute of Environmental Science and Research (ESR) Ltd., where they are collated, analysed and reported on behalf of the Ministry of Health. Further information about notifiable diseases can be found in the 2009 Annual Surveillance Report (Population and Environmental Health Group (ESR), 2010).

2.1.2 Laboratory-Based Surveillance

The reference laboratories at ESR maintain databases of laboratory results for notifiable diseases.

The number of laboratory-reported salmonellosis cases has, until recently, always exceeded the number of notifications. The implementation of integration processes in 2004 for notifications and laboratory results at ESR has addressed this problem.

2.1.3 Ministry of Health (MoH)

MoH collates national data on patients admitted and discharged from publicly funded hospitals. These data are stored as part of the National Minimum Dataset (NMDS). Cases are assigned disease codes using the tenth revision of the International Classification of Diseases (ICD-10) coding system. Up to 99 diagnostic, procedure, and accident codes may be assigned to each admission. The first of these is the principal or primary diagnosis, which is the condition that actually led to admission. This may differ from the underlying diagnosis.

Hospital admission data include repeated admissions for patients with chronic notifiable diseases (e.g. tuberculosis) or diseases which have long-term health impacts (e.g. meningococcal disease). For some diseases, the criteria for notification (clinical and laboratory or epidemiological evidence) do not match those required for diagnostic coding. For these reasons hospitalisation numbers and notifications may differ. In this report hospitalisations, including readmissions, have been reported for all primary disease. For the disease sequelae (GBS and HUS) there is potential

for multiple readmissions. Readmissions within the calendar year were removed and reported case numbers represent unique cases, rather than total admissions.

2.1.4 Outbreak Surveillance

ESR has operated an outbreak surveillance system in EpiSurv since mid-1997. This enables PHUs to record and report outbreaks for national reporting and analysis. In particular, it should be noted that not all cases associated with outbreaks are recorded as individual cases of notifiable disease in EpiSurv. The terms 'setting' and 'suspected vehicle' are both used in outbreak reporting to describe likely implicated sources found in epidemiological or environmental investigations. More information about outbreak reporting system can be found in the 2009 Disease Outbreak Report (Population and Environmental Health Group (ESR), 2010).

2.1.5 Statistics New Zealand

Data from the Statistics New Zealand website www.stats.govt.nz was used to calculate notification and hospitalisation population rates of disease. See analytical methods section for further details.

2.1.6 NZFSA project reports and publications

NZFSA project reports, prepared by ESR or other providers, and publications from the general literature were used to provide specific contextual information on the prevalence of selected pathogens in specific food types.

2.1.7 Risk attribution

Information from a NZFSA project on risk ranking was used to estimate the proportion of disease due to specific pathogens that can be attributed to transmission by food (Cressey and Lake, 2005). Attributable proportions were determined by expert consultation, using a modified double-pass Delphi, with a facilitated discussion between passes. Each expert was asked to provide a minimum ('at least'), a most likely and a maximum ('not more than') estimate of the proportion of a number of microbial diseases that were due to transmission by food. Estimates presented in the current report are mean values from the second pass.

2.2 Analytical Methods

Key analytical methods used include:

2.2.1 Dates

Notification and outbreak data contained in this report are based on information recorded in EpiSurv as at 12 February 2010. Changes made to EpiSurv data by PHU staff after this date will not be reflected in this report. Consequently, future analyses of these data may produce revised results. Disease numbers are reported according to the date of notification. Laboratory results are reported according to the date the specimen was received.

2.2.2 Data used for calculating rates of disease

All population rates use Statistics New Zealand mid-year population estimates as at 30 June 2009 and are crude rates unless otherwise stated. Rates have not been calculated where there are fewer than five notified cases or hospitalisations in any category. Calculating rates from fewer than five cases produces unstable rates.

2.2.3 Geographical breakdown

This report provides rates for current District Health Boards (DHBs). The DHB populations have been derived from the Statistics New Zealand mid-year population estimates for Territorial Authorities in New Zealand.

2.2.4 Map classification scheme

The maps classification for the disease rates is quantiles i.e. the data have been divided into three groups (tertiles) containing equal numbers of DHBs. The darkest colour represents the highest rates and the lightest colour the lowest rates. The grey colour shows where there are insufficient data to calculate a rate (less than 5 cases).

2.2.5 Risk factors and source of infection

For many diseases an analysis of exposure to risk factors for the cases is reported. The risk factor questions on the EpiSurv case report forms are those that are currently known for that disease. Often more than one risk factor is reported for each case. The high number of unknown outcomes associated with the risk factors should be noted.

The reporting of exposure to a risk factor does not imply that this was the source of the infection.

2.2.6 Statistical tests

Confidence intervals have been calculated for the disease rates and displayed on the graphs. The historical mean is calculated from the previous three years data (2006-2008).

2.3 Interpreting Data

Data in this report may differ from those published in other reports depending on:

- the date of extraction of data
- the date used to aggregate data (e.g. date reported or date of onset of illness)
- filters used to extract the data

The information in this report shows disease trends by age group, sex, and place of residence (District Health Board).

Because of the low numbers of cases for some conditions and age groups, etc. the rates calculated in this report may be highly variable from year to year and it is necessary to interpret trends with caution.

3 THE ACUTE GASTROINTESTINAL ILLNESS (AGI) STUDY

The Acute Gastrointestinal Illness (AGI) Study is a set of three linked surveys, with the following objectives:

- To determine the magnitude and distribution of self reported AGI in the New Zealand population;
- To estimate the burden of disease associated with AGI;
- To describe and estimate the magnitude of under-ascertainment of AGI at each stage in the national communicable disease surveillance process; and,
- To identify modifiable factors affecting under-ascertainment that, if altered, could reduce case loss throughout the AGI component of the surveillance system.

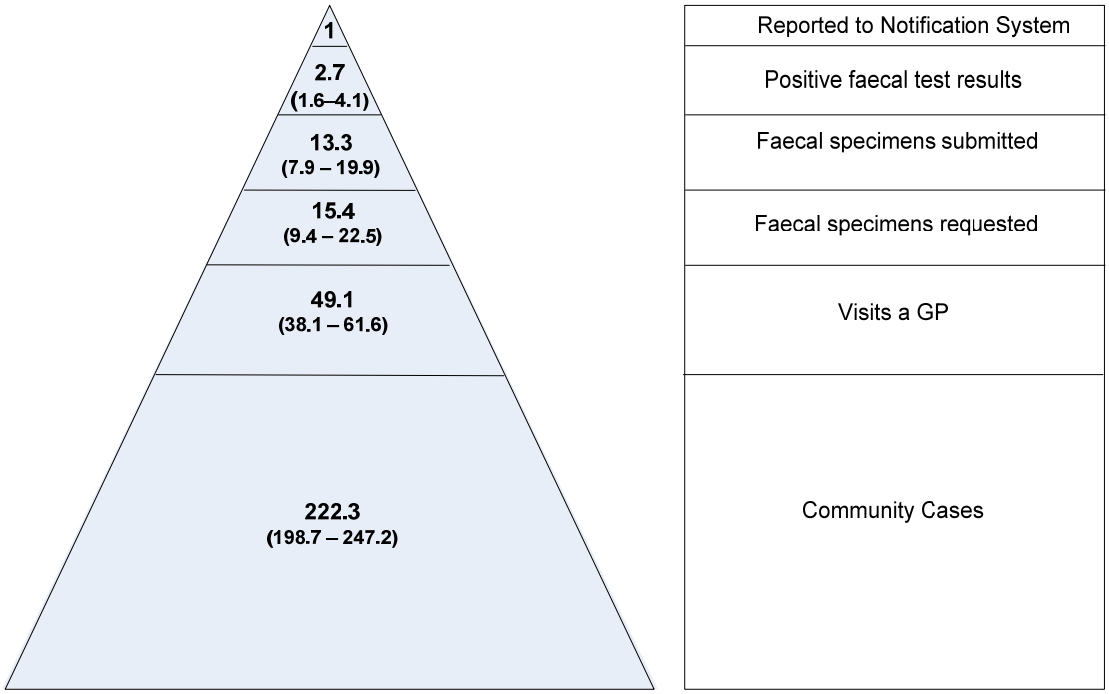
The three study elements were completed during 2005-2007 and each has been reported separately (available from the NZFSA website: http://www.nzfsa.govt.nz/science/research-projects/index.htm):

- Community study: a twelve month telephone survey conducted from February 2006 – January 2007 and reported as "Acute Gastrointestinal Illness (AGI) Study: Community Survey" (Adlam *et al.*, 2007),
- General practice study: a nationwide incidence study conducted over seven weeks from May – July 2006, using selected practices via a computer network practice management system, supplemented by a postal survey conducted in July 2006. This study has been reported as "Acute Gastrointestinal Illness (AGI) Study: General Practice Study" (Perera and Adlam, 2007), and
- Laboratory study: a postal survey of 45 community and hospital laboratories conducted in June 2006, and reported as "Acute Gastrointestinal Illness (AGI) Study: Laboratory Survey" (King *et al.*, 2007).

The results from the Community survey indicated that the incidence of AGI was 1.1 per person year, representing 4.66 million cases in New Zealand in one year. These illnesses are caused by microbial hazards that may be transmitted by a number of routes, including foods. However, at this stage it is not possible to identify the total fraction of AGI caused by foodborne transmission.

A final report amalgamated results from the three studies was produced to construct a reporting pyramid for AGI in New Zealand, as shown in Figure 1 (Lake *et al.*, 2007). It is important to recognise that this pyramid applies to AGI in its entirety, and cannot be applied to AGI caused by individual pathogens, which may have quite different ratios.

Figure 1: Reporting pyramid (areas to scale) for New Zealand showing ratios of cases in the community, general practice, and clinical laboratory levels relative to notifiable diseases, 2006 (mean, 5th and 95th percentiles)



The reporting pyramid is constructed from data reported from the community survey (Adlam *et al.*, 2007); GP survey (Perera and Adlam, 2007); and laboratory survey (King *et al.*, 2007).

Note that not all positive faecal test results will be for diseases that are notifiable.

4 REPORTING

4.1 Reporting Against Targets

In 2007, NZFSA established three performance goals for potentially foodborne illnesses.

4.1.1 Performance goals

- Campylobacteriosis: 50% reduction in foodborne component after a period of 5 years
- Salmonellosis: 30% reduction in foodborne component after a period of 5 years
- Listeriosis: No increase in the foodborne component with increasing range of foods available (including raw milk cheeses).

4.1.2 Rationale

The above diseases include the two most commonly notified, potentially foodborne illnesses in New Zealand plus listeriosis, one of the most severe. This selection is based, in part, on the ESR foodborne illness attribution work which identified campylobacteriosis and listeriosis as creating the highest human health burden within New Zealand (Cressey and Lake, 2007). The inclusion of salmonellosis will also allow for New Zealand comparability with US and UK monitoring programmes. For the period 2004-2007 there were approximately 13 600 notified cases of campylobacteriosis, 1 150 of salmonellosis and 23 of listeriosis annually in New Zealand. Foodborne illness due to VTEC/STEC infections is not included as there are only about 10 cases per year that could be attributable to foodborne sources. Norovirus is not incorporated at this stage because of the large fluctuations that occur in annual statistics (norovirus infection only became a notifiable disease in December 2007) and, for most cases, the causality (e.g. person-to-person) is likely to be outside of the influence of NZFSA.

The performance goals for the foodborne diseases have been determined by the NZFSA Board and aligned with expectations arising from current regulatory priorities and programmes (e.g. the NZFSA *Campylobacter* Risk Management Strategy 2008-2011). Notwithstanding yearly variations, a robust performance monitoring system should be able to measure trends in risk reduction over time e.g. for *Campylobacter*.

4.1.3 Methodology, tools and reporting

Historical baseline data on the number of reported cases of the targeted foodborne diseases are available and NZFSA is supporting projects to increase the quality of data. The source of the data is the *Notifiable and Other Diseases in New Zealand Annual Report,* by ESR. The NZFSA Science Group is funding active surveillance projects that will provide primary information on food attribution such as the advanced attribution study conducted by Massey University and Mid-Central Health within the Manawatu.

The measurement will be adjusted for the proportion of cases reported as having travelled overseas during the likely incubation period. It will be adjusted also for the proportion of disease estimated to be due to foodborne transmission.

The annual incidence of campylobacteriosis and salmonellosis will be reported in terms of calendar year totals of cases per 100 000-people (*Notifiable and Other Diseases in New Zealand Annual Report,* ESR). This allows for demographic changes within the New Zealand population to

be appropriately captured. The proportion of cases acquired abroad will be estimated through the EpiSurv programme administered by ESR and MoH[1]. Estimates of the foodborne proportion of selected communicable diseases have been determined by expert elicitation and are approximately 0.6, 0.6 and 0.9 respectively for campylobacteriosis, salmonellosis and listeriosis.

From year to year, fluctuations in disease rates may occur due to modifications in clinical, laboratory and notification practices as well as changes in food exposure. These will be highlighted and corrected for where possible.

4.1.4 Campylobacteriosis

4.1.4.1 Performance goal

- 50% reduction in reported annual incidence of foodborne campylobacteriosis after five years

4.1.4.2 Measurement

Annual (calendar year) number (per 100 000 mid-year population estimate) of notified cases of human campylobacteriosis, with the baseline year being average of 2004-2007. The measurement will be adjusted for the proportion of cases reported as having travelled overseas during likely incubation period; and for the proportion of disease estimated to be due to foodborne transmission (Table 4).

Table 4: Estimated proportion of foodborne campylobacteriosis for 2009

	Cases	Proportion (%)	Rate (per 100 000, mid year estimated population)
Total notified	7 176		166.3
Estimated not travelled overseas	6 671	93.0	154.6
Estimated foodborne transmission proportion	3 836	57.5 (37.1 – 69.6)*	88.9 (57.3 – 107.6)#

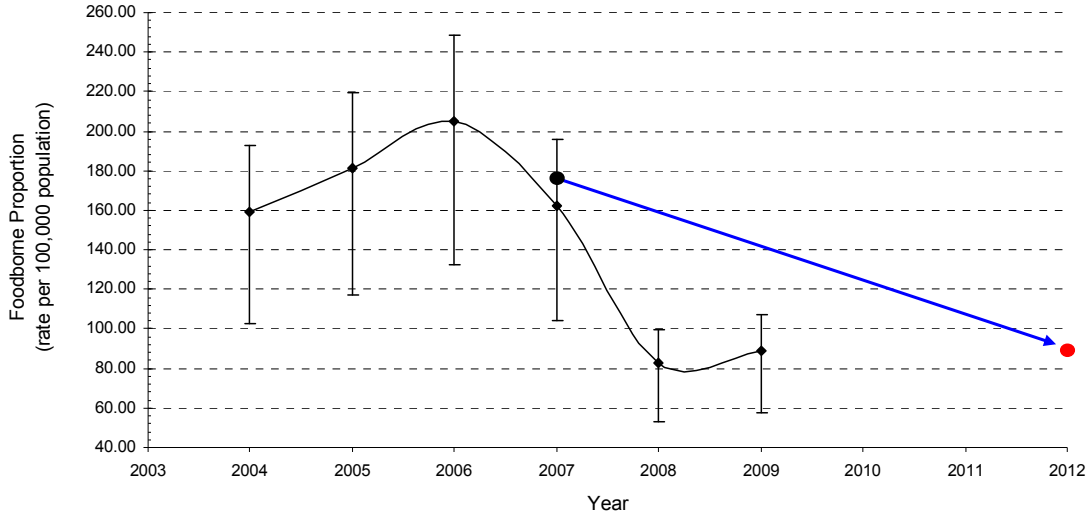
* Most likely (Minimum – Maximum) estimates of proportion foodborne, from expert consultation
# Most likely (Minimum – Maximum) estimates of foodborne rate

4.1.4.3 Presentation

The trend in relative rates (and ranges) compared with the baseline and five year goal is shown in Figure 2.

[1] Assuming that the cases for which travel information was provided are representative of all cases, a Poisson distribution can be used to estimate the total number of potentially travel related cases

Figure 2: Foodborne proportion of campylobacteriosis



The blue arrowed line represents the trend line from the baseline year (average of 2004-2007) to the five year target (red dot)

4.1.5 Salmonellosis

4.1.5.1 Performance target

- 30% reduction in reported annual incidence of foodborne salmonellosis after five years

4.1.5.2 Measurement

Annual (calendar year) number (per 100 000 mid year population estimate) of notified cases of human salmonellosis, with the baseline being 2004-2007. The measurement will be adjusted for the proportion of cases reported as having travelled overseas during likely incubation period; and for the proportion of disease estimated to be due to foodborne transmission (Table 5).

Table 5: Estimated proportion of foodborne salmonellosis for 2009

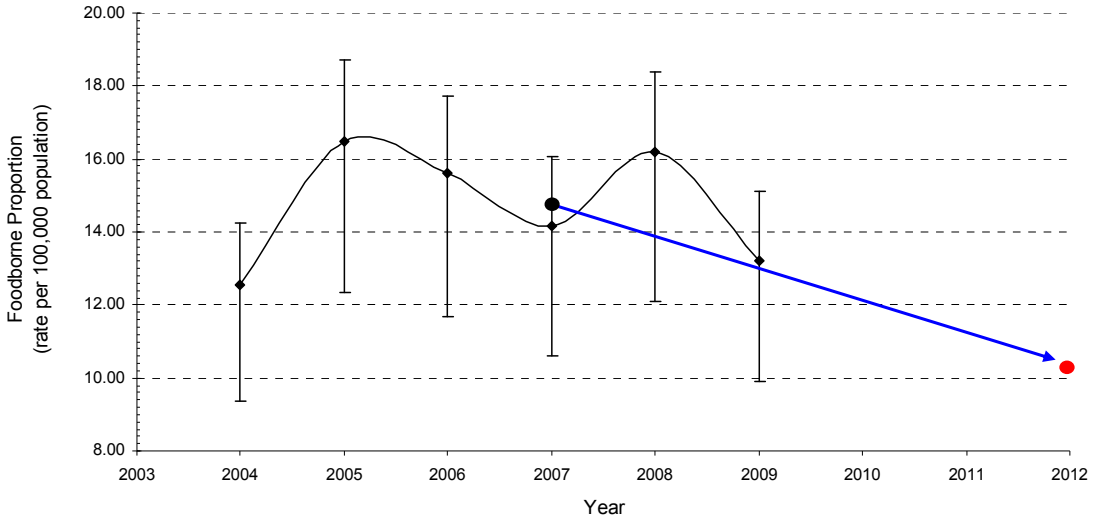
	Cases	Proportion (%)	Rate (per 100 000, mid year estimated population)
Total notified cases	1 129		26.2
Estimated not travelled overseas	943	83.6	21.8
Estimated foodborne transmission proportion	572	60.7 (45.4 -68.9)*	13.2 (9.9 – 15.1)#

* Most likely (Minimum – Maximum) estimates of proportion foodborne, from expert consultation
\# Most likely (Minimum – Maximum) estimates of foodborne rate

4.1.5.3 Presentation

The trend in relative rates (and ranges) compared with the baseline and five year goal is shown in Figure 3.

Figure 3: Foodborne proportion of salmonellosis



The blue arrowed line represents the trend line from the baseline year (average of 2004-2007) to the five year target (red dot)

4.1.6 Listeriosis

4.1.6.1 Performance target

- No increase in reported annual incidence of foodborne listeriosis after five years

4.1.6.2 Measurement

Annual (calendar year) number (per 100 000 population) of notified cases of human listeriosis, with the baseline being 2005-2008. The measurement will be adjusted for the proportion of cases reported as having travelled overseas during likely incubation period; and for the proportion of disease estimated to be due to foodborne transmission (Table 6).

Table 6: Estimated proportion of foodborne listeriosis for 2009

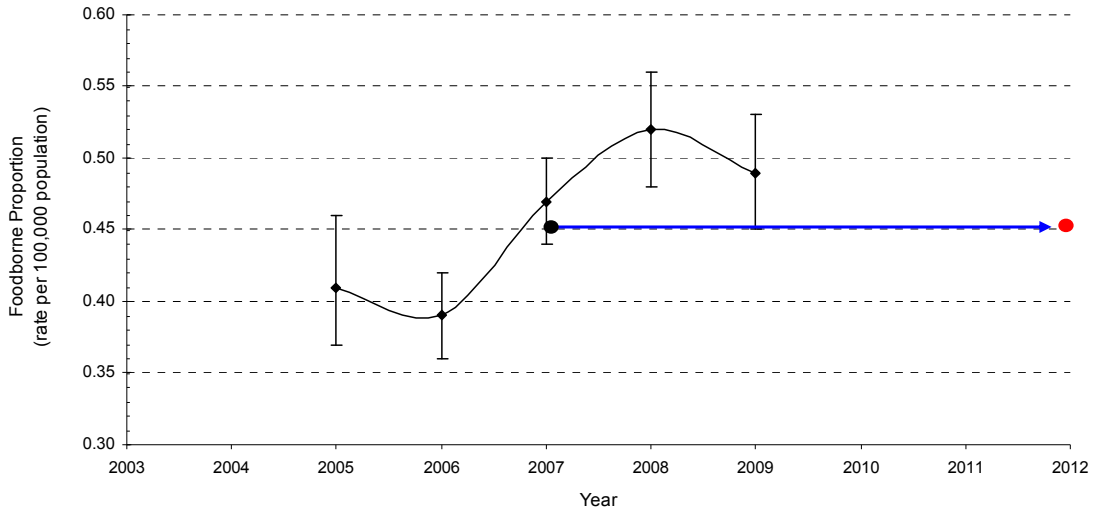
	Cases	Proportion (%)	Rate (per 100 000, mid year estimated population)
Total notified cases	28		0.65
Estimated not travelled overseas	25	90.9	0.58
Estimated foodborne transmission proportion	21	84.9 (78.4 – 92.1)*	0.49 (0.45 – 0.53)#

* Most likely (Minimum – Maximum) estimates of proportion foodborne, from expert consultation
\# Most likely (Minimum – Maximum) estimates of foodborne rate

4.1.6.3 Presentation

The trend in relative rates (and ranges) compared with the baseline and five year goal is shown in Figure 4.

Figure 4: Foodborne proportion of listeriosis



The blue arrowed line represents the trend line from the baseline year (average of 2004-2007) to the five year target (red dot)

4.2 Incidence and Severity of Selected Foodborne Diseases

This section includes a summary for each potentially foodborne condition. For conditions with sufficient numbers (approximately 100 cases or more per year) a full analysis, drawn from notification, hospitalisation, mortality, and laboratory data, has been carried out. For diseases with a small number of cases a more limited analysis has been carried out.

These data are followed by contextual information on the foodborne proportion of the overall incidence of illness. This section will include information on the following topics, where available:

- Statement of estimated foodborne percentage and range provided by an expert elicitation process conducted in 2004-2005. Note that these estimates are only available for some of the illnesses included in this report;
- Statement of estimated foodborne percentage and range for any specific foods provided by the same expert elicitation process;
- Information on pathogen typing (principally from data generated by ESR's Enteric Reference Laboratory), where it is available and informative about foodborne disease;
- Comments on specific food related incidents or outbreaks of the disease that were reported to the notification system during the calendar year;
- Studies on foodborne attribution for the specific disease conducted or published during the calendar year;
- Information on the prevalence of the chemical or microbial hazard in particular foods as a result of surveys conducted during the calendar year; and,
- Regulatory or other risk management actions in New Zealand that might be expected to affect the foodborne disease data.

4.3 *Bacillus cereus* Intoxication

4.3.1 Case definition

Clinical description:	Gastroenteritis where either vomiting or profuse watery diarrhoea dominate
Laboratory test for diagnosis:	Isolation of ≥10^3/g *B. cereus* from a clinical specimen or ≥10^4 *B. cereus* from leftover food or detection of diarrhoeal toxin in a faecal sample
Case classification:	
Probable	A clinically compatible illness that is either a contact of a confirmed case of the same disease, or has had contact with the same common source i.e., is part of an identified common source outbreak
Confirmed	A clinically compatible illness that is laboratory confirmed

4.3.2 *Bacillus cereus* intoxication cases reported in 2009 by data source

During 2009, no notifications of *Bacillus cereus* intoxication were reported in EpiSurv.

The ICD-10 code A05.4 was used to extract *Bacillus cereus* intoxication hospitalisation data from the MoH NMDS database. There were no hospital admissions recorded in 2009 with *Bacillus cereus* intoxication as a primary or other relevant diagnosis.

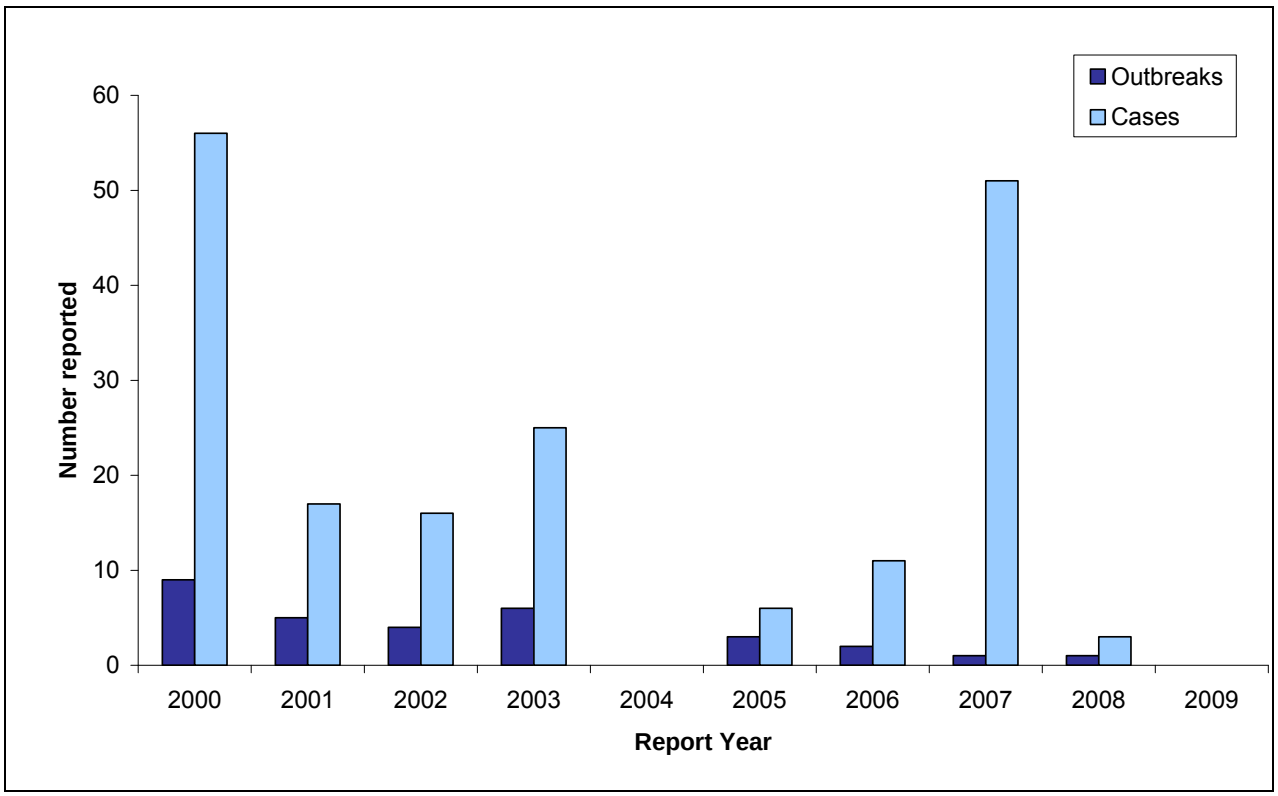
Expert consultation estimated that 97% (minimum = 90%, maximum = 99%) of *Bacillus cereus* intoxication will be due to foodborne transmission. The expert consultation also estimated that approximately 60% of the foodborne transmission would be due to consumption of rice.

4.3.3 Outbreaks reported as caused by *Bacillus cereus*

No *Bacillus cereus* outbreaks were reported in EpiSurv during 2009.

From 2004 to 2009, fewer outbreaks were reported each year in EpiSurv than in any of the four years prior to 2004 (Figure 5).

Figure 5: Foodborne *Bacillus cereus* outbreaks and associated cases reported by year, 2000–2009



4.3.3.1 Laboratory investigation of samples from suspected foodborne outbreaks

During investigations of suspected foodborne illness outbreaks by ESR's Public Health Laboratory, elevated levels of *Bacillus cereus* (>10^5 CFU/g) were isolated from food samples (butter chicken and rice) associated with one investigation. Diarrhoeal toxin was also detected in the faeces of one case from this investigation. In a second investigation, diarrhoeal toxin was detected in a faecal sample, but only marginal to low concentrations of *Bacillus cereus* (<10^3) were found in the implicated food (stuffed roast chicken).

4.3.4 Recent surveys

Nil.

4.3.5 Relevant New Zealand studies and publications

Nil.

4.3.6 Relevant regulatory developments

Nil.

4.4 Campylobacteriosis

Summary data for campylobacteriosis in 2009 are given in Table 7.

Table 7: Summary surveillance data for campylobacteriosis, 2009

Parameter	Value in 2009	Section reference
Number of cases	7 176	4.4.2
Rate (per 100 000)	166.3	4.4.2
Hospitalisations (%)	574 (8.0%)	4.4.2
Deaths (%)	0 (0%)	4.4.2
Estimated travel-related cases (%)	505 (7.0%)	4.4.3.6
Estimated food-related cases (%)*	3 836 (57.5%)	4.4.2

* For estimation of food-related cases it was assumed that the proportions derived from expert consultation would exclude travel-related cases

4.4.1 Case definition

Clinical description: An illness of variable severity with symptoms of abdominal pain, fever and diarrhoea, and often bloody stools

Laboratory test for diagnosis: Isolation of *Campylobacter* from a clinical specimen

Case classification:

Probable: A clinically compatible illness that is either a contact of a confirmed case of the same disease, or has had contact with the same common source i.e., is part of an identified common source outbreak

Confirmed: A clinically compatible illness that is laboratory confirmed

4.4.2 Campylobacteriosis cases reported in 2009 by data source

During 2009, 7 176 notifications (166.3 per 100 000 population) of campylobacteriosis were reported in EpiSurv.

The ICD-10 code A04.5 was used to extract campylobacteriosis hospitalisation data from the MoH NMDS database. Of the 574 hospital admissions (13.3 admissions per 100 000 population) recorded in 2009, 473 were reported with campylobacteriosis as the primary diagnosis and 101 with campylobacteriosis as another relevant diagnosis.

No deaths due to campylobacteriosis were recorded in EpiSurv in 2009.

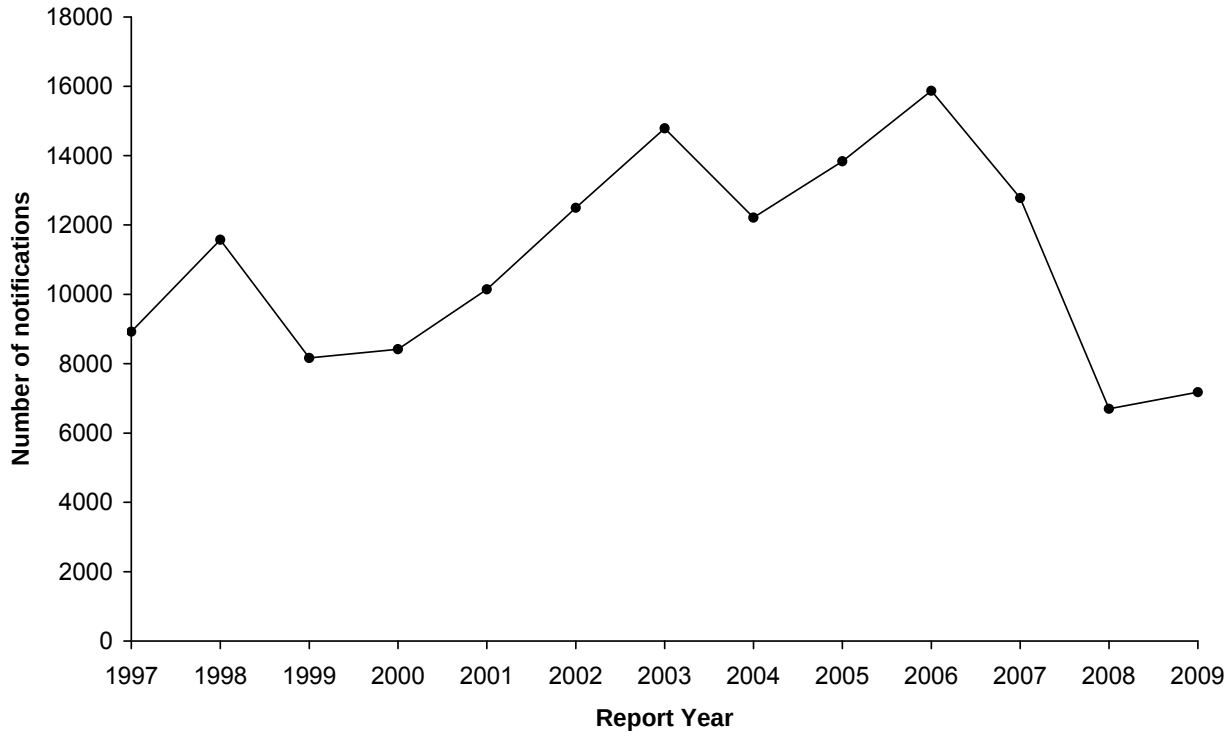
It has been estimated by expert consultation that 57.5% (minimum = 37%, maximum = 70%) of campylobacteriosis incidence is due to foodborne transmission. It was further estimated that 53% of foodborne transmission would be due to transmission via poultry.

4.4.3 Notifiable disease data

4.4.3.1 Annual notification trend

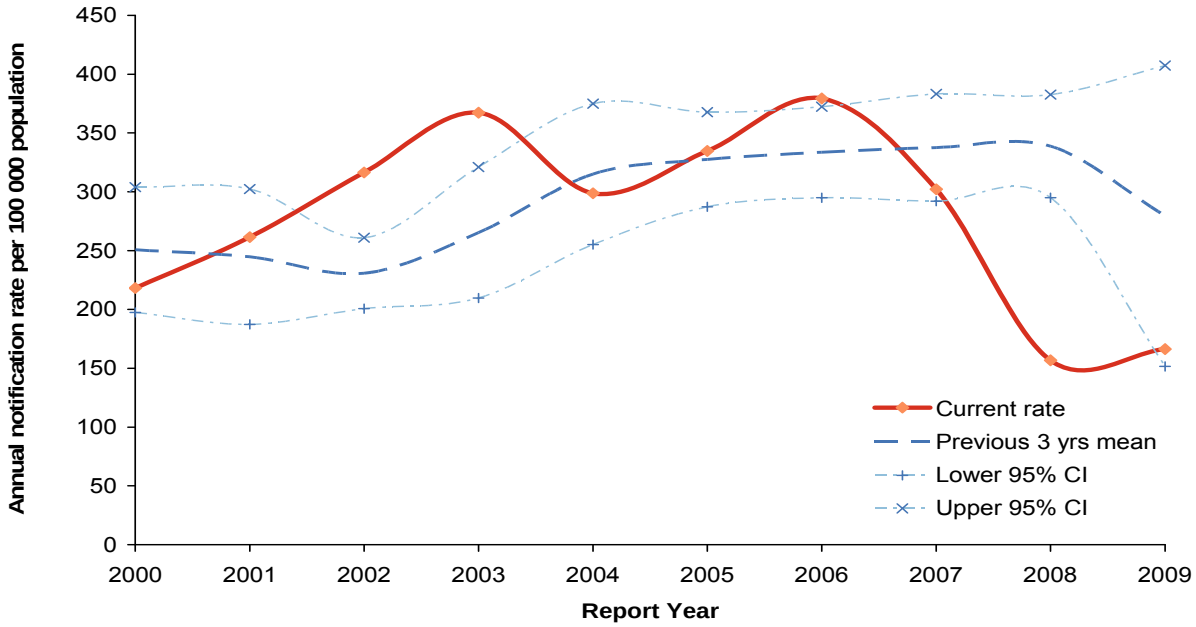
The number of campylobacteriosis notifications reported each year generally increased from 1996, with the highest number recorded in 2006 (15 873 cases). Since 2006, there has been a significant decrease in the number of cases reported (Figure 6).

Figure 6: Campylobacteriosis notifications by year, 1997-2009



The campylobacteriosis annual rate trend (Figure 7) was very similar to the corresponding annual notification trend; with a general increase in the notification rate observed over the period 2000-2006 followed by a sudden reduction in 2007 to 2009.

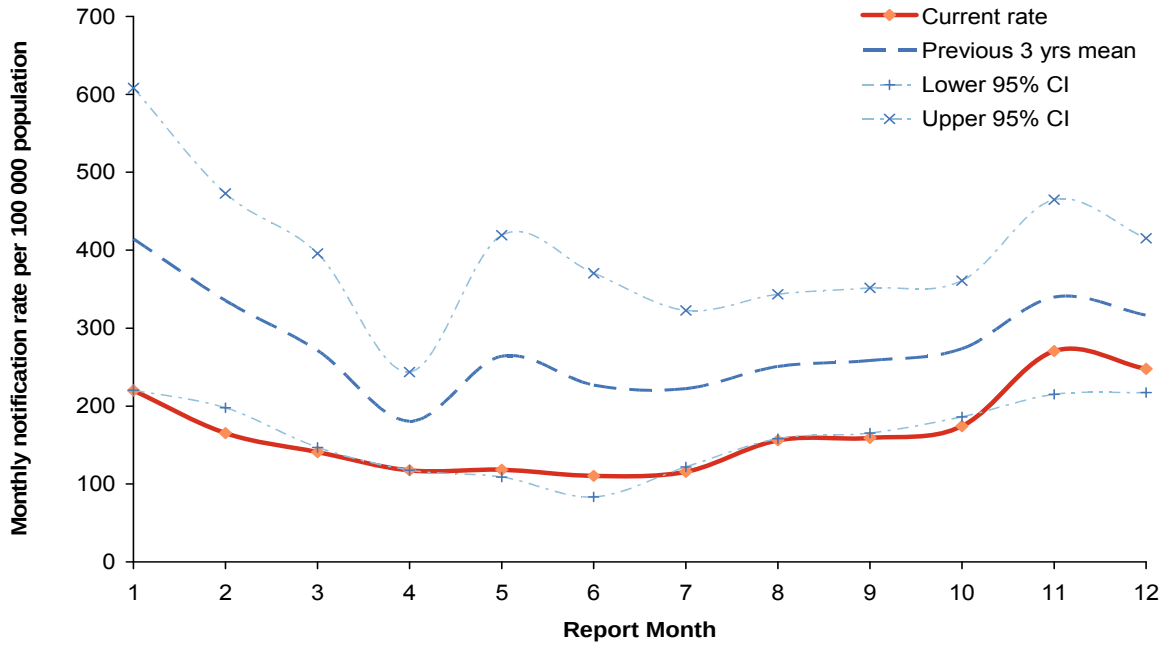
Figure 7: Campylobacteriosis notification rate by year, 2000-2009



4.4.3.2 Seasonality

The number of notified cases of campylobacteriosis per 100 000 population by month for 2009 is shown in Figure 8. The pattern in 2009 is similar to previous years, highly seasonal with a summer peak and winter trough. The lowest monthly campylobacteriosis notification total for 2009 was for the month of June with 397 notifications and the highest was for the month of November when 973 cases were notified.

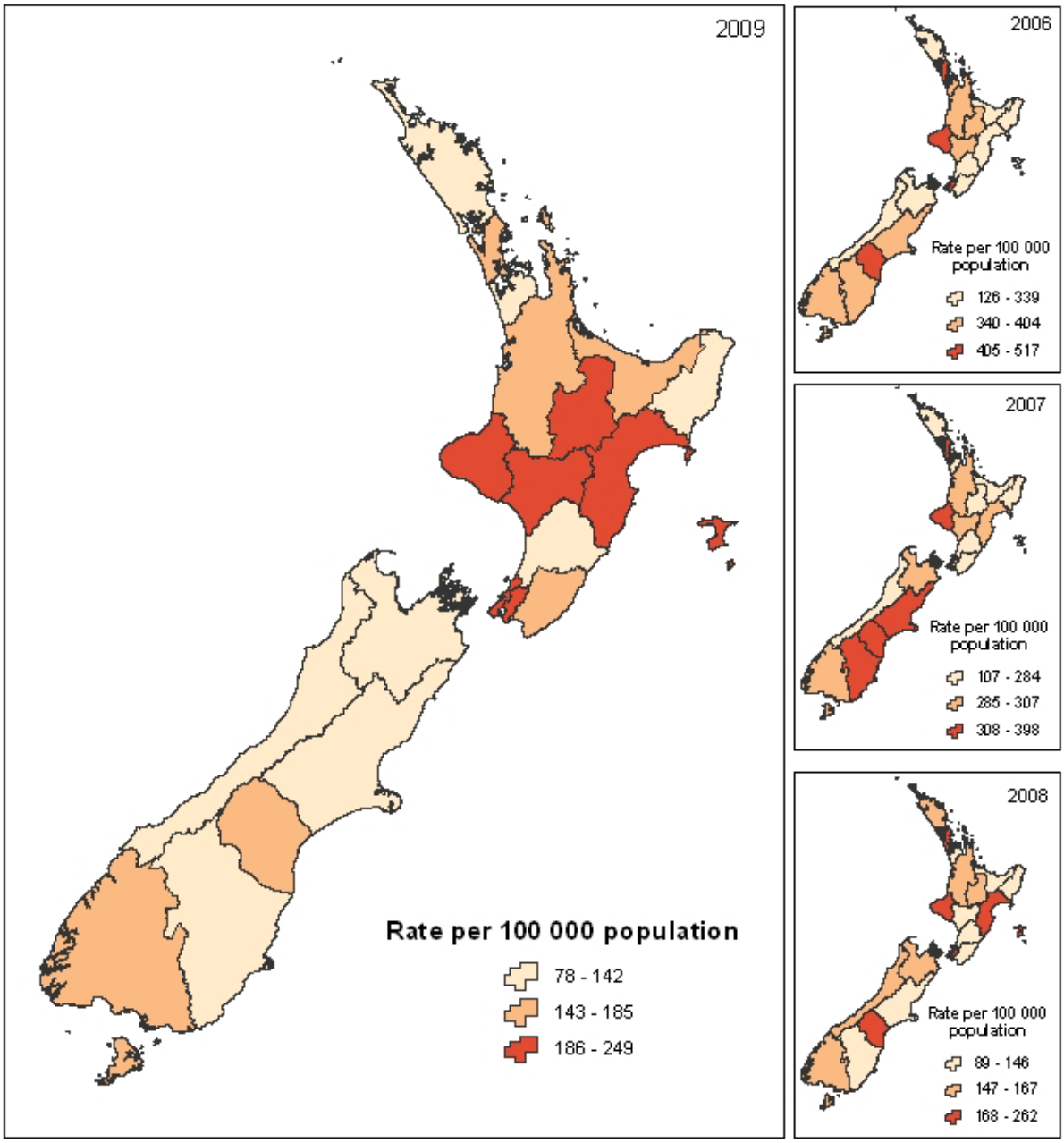
Figure 8: Campylobacteriosis monthly rate (annualised) for 2009



4.4.3.3 Geographic distribution of campylobacteriosis notifications

Campylobacteriosis rates varied throughout the country as shown in Figure 9. The highest rates were reported in Hutt Valley (248.8 per 100 000 population, 355 cases) and Capital and Coast (240.2 per 100 000, 692 cases) DHBs. The lowest rates were reported in Tairawhiti (77.9 per 100 000, 36 cases) and Canterbury (108.6 per 100 000, 545 cases) DHBs. Taranaki DHB has been in the highest quantile of campylobacteriosis notification rates for each of the last four years.

Figure 9: Geographic distribution of campylobacteriosis notifications, 2006-2009



4.4.3.4 Age and sex distribution of campylobacteriosis cases

In 2009, the number and rate of notifications and hospitalisations for campylobacteriosis were higher in males than in females (Table 8).

Table 8: Campylobacteriosis cases by sex, 2009

Sex	EpiSurv notifications		Morbidity[a]		Deaths recorded in EpiSurv
	No.	Rate[b]	No.	Rate[b]	No.
Male	3 976	187.8	291	13.7	
Female	3 119	141.9	283	12.9	
Unknown	81				
Total	7 176	166.3	574	13.3	

[a] MoH morbidity data for hospital admissions
[b] per 100 000 of population

The highest age-specific notification rate for campylobacteriosis in 2009 occurred for children aged 1 to 4 years (337.4 per 100 000 population, 818 cases) and children aged less than one year (247.3 per 100 000, 156 cases). The hospitalisation rate for the 70+ years age group was more than double that reported in any other age group (Table 9).

Table 9: Campylobacteriosis cases by age group, 2009

Age group	EpiSurv notifications		Morbidity[a]		Deaths recorded in EpiSurv
	No.	Rate[b]	No.	Rate[b]	No.
<1	156	247.3	10	15.9	
1 to 4	818	337.4	30	12.4	
5 to 9	370	128.4	16	5.6	
10 to 14	325	109.2	24	8.1	
15 to 19	495	153.2	42	13.0	
20 to 29	1 262	215.7	100	17.1	
30 to 39	838	145.4	57	9.9	
40 to 49	887	139.7	45	7.1	
50 to 59	773	145.5	49	9.2	
60 to 69	666	169.5	53	13.5	
70+	563	147.8	148	38.9	
Unknown	23				
Total	7 176	166.3	574	13.3	

[a] MoH morbidity data for hospital admissions
[b] per 100 000 of population

4.4.3.5 Risk factors reported

The risk factors recorded for campylobacteriosis in 2009 are shown in Table 10. The most common risk factors reported were consumption of food from retail premises (44.4%) and contact with farm animals (43.2%).

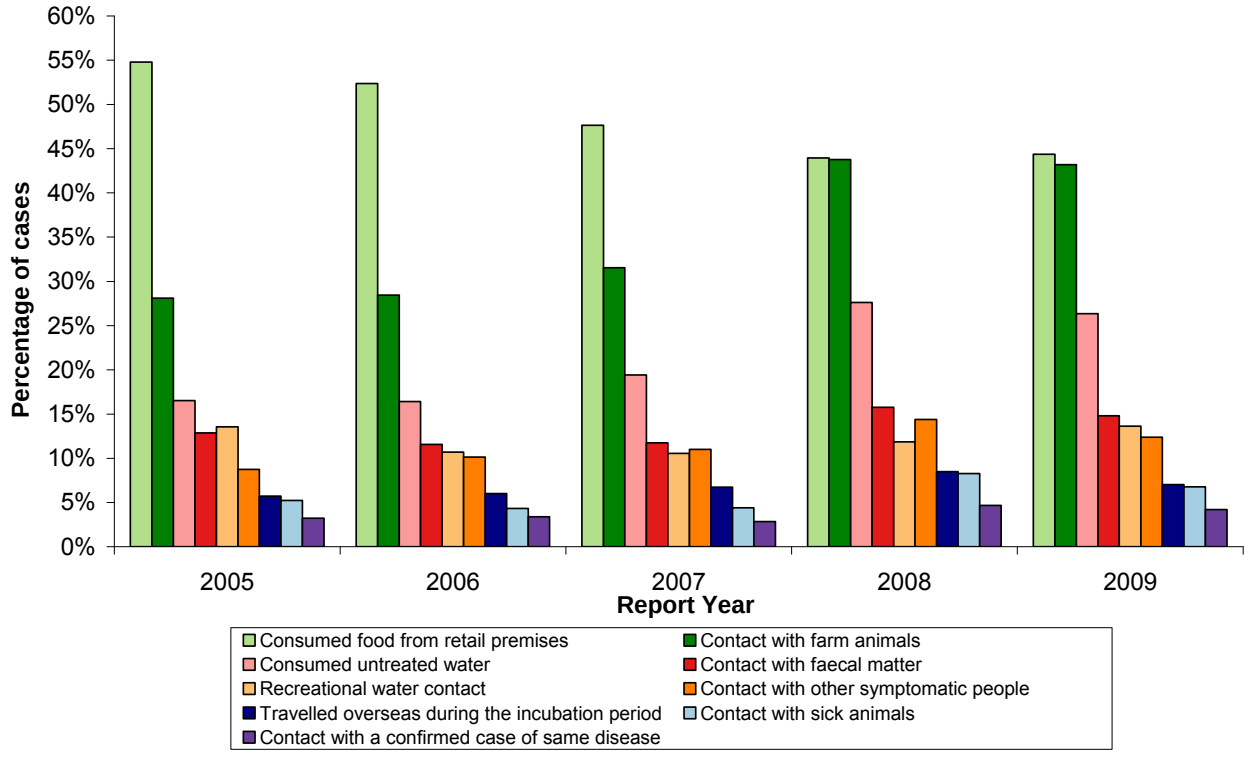
Table 10: Exposure to risk factors associated with campylobacteriosis, 2009

	Notifications			
Risk Factor	**Yes**	**No**	**Unknown**	**%[a]**
Consumed food from retail premises	822	1 031	5 323	44.4
Contact with farm animals	842	1 107	5 227	43.2
Consumed untreated water	420	1 173	5 583	26.4
Contact with faecal matter	255	1 467	5 454	14.8
Recreational water contact	234	1 484	5 458	13.6
Contact with other symptomatic people	220	1 559	5 397	12.4
Travelled overseas during the incubation period	154	2 035	4 987	7.0
Contact with sick animals	112	1 541	5 523	6.8
Contact with a confirmed case of same disease	71	1 622	5 483	4.2

[a]Percentage refers to the cases that answered "yes" out of the total number of cases for which this information was supplied. Cases may have more than one risk factor recorded.

Between 2005 and 2009, consumption of food from retail premises, contact with farm animals, and consumption of untreated water were consistently the most commonly reported risk factors for campylobacteriosis. There has been a decrease in the percentage of cases that reported consuming food from retail premises and this risk factor is now reported by a similar percentage to those who report contact with farm animals (Figure 10).

Figure 10: Campylobacteriosis risk factors by percentage of cases and year, 2005-2009



4.4.3.6 Estimate of travel-related cases

For cases where information on travel was provided in 2009, 7.0% (95%CI 6.0-8.2%) had travelled overseas during the incubation period. Assuming that the cases for which travel information was provided were representative of all campylobacteriosis cases, a Poisson distribution can be used to estimate the total number of potentially travel related cases of campylobacteriosis in 2009. The resultant distribution has a mean of 505 cases (95% CI 417-600).

If data from the last four years are considered the estimated proportion of cases travelling overseas within the incubation period of the organism is 6.8% (95% CI 6.4-7.2%). The proportion of travel-associated cases in 2009 was lower than in 2008, but close to the four year average.

4.4.4 Outbreaks reported as caused by *Campylobacter* spp.

In this section only *Campylobacter* spp. outbreaks with a suspected or known foodborne source are included unless otherwise stated.

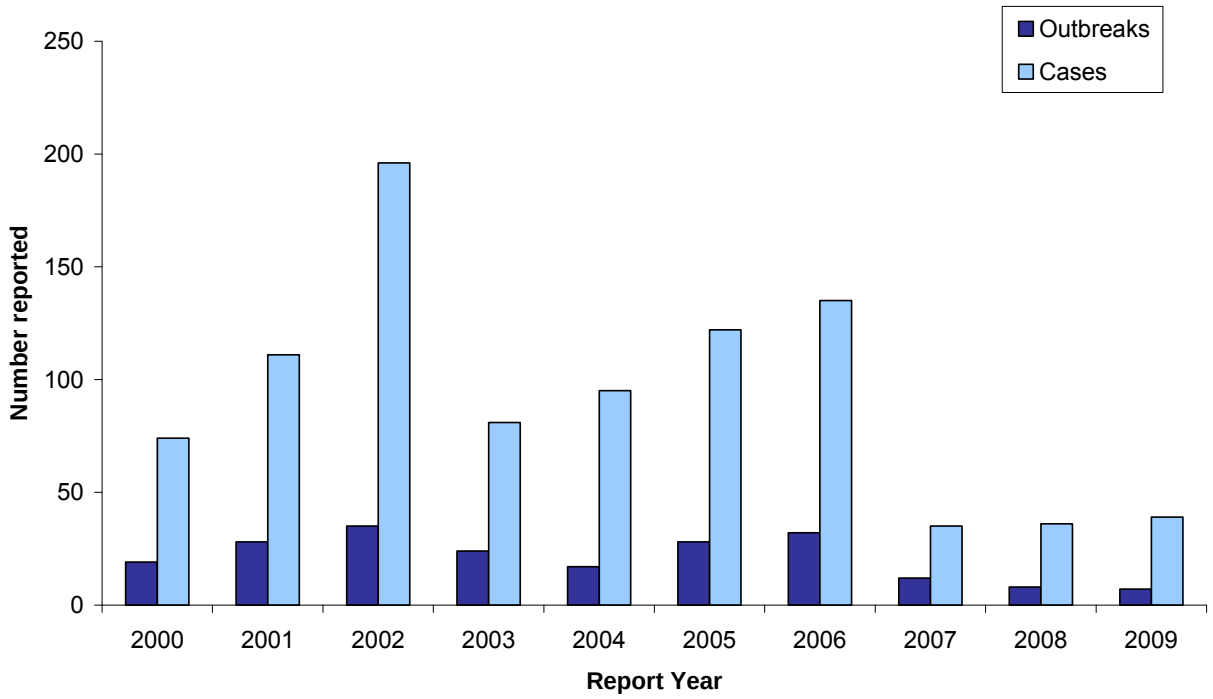
In 2009, seven (58.3%) of the *Campylobacter* outbreaks and 39 (60.0%) of the associated cases were reported as foodborne (Table 11). *Campylobacter* outbreaks accounted for 1.9% (12/639) of all outbreaks and 0.6% (65/10 736) of all associated cases.

Table 11: *Campylobacter* spp. outbreaks reported, 2009

Measure (No.)	Foodborne *Campylobacter* spp. outbreaks	All *Campylobacter* spp. outbreaks
Outbreaks	7	12
Cases	39	65
Hospitalised cases	0	5

The number of foodborne *Campylobacter* outbreaks and associated cases increased from 17 outbreaks (95 cases) in 2004 to 32 outbreaks (135 cases) in 2006. In 2007 the number of foodborne *Campylobacter* outbreaks decreased markedly to 12 outbreaks and in 2009 the lowest number of outbreaks (7) was reported of any of the 10 years, 2000-2009 (Figure 11).

Figure 11: Foodborne *Campylobacter* spp. outbreaks and associated cases reported by year, 2000-2009



4.4.4.1 Details of food-associated outbreaks

Table 12 contains details of the seven food–associated *Campylobacter* spp. outbreaks reported in 2009.

Table 12: Details of food-associated *Campylobacter* spp. outbreaks, 2009

Public Health Unit (Month)	Suspected vehicle	Setting	Number ill	Confirmation
Auckland (August)	Chicken	Home	2C, 1P	6
Manawatu (May)	Chicken and beef liver	Hostel	2C, 1P	1
Manawatu (May)	Unpasteurised milk	Home	2C	6
Manawatu (December)	Chicken	Hospital (continuing care), Rest home	2C, 6P	7
Northland (August)	Unpasteurised milk	Farm	4C, 12P	1, 2, 5
Southland (June)	Bangers and mash or lambs fry	Restaurant/Café	2C, 1P	6
Wellington (April)	Chicken liver pate	Restaurant/Café	1C, 3P	2

C = confirmed, P = probable
Confirmation:
1 = Environmental investigation – identified critical control point failures linked to implicated source
2 = Epidemiological – case had history of exposure to implicated source
3 = Epidemiological – case control or cohort study showed elevated risk for cases to implicated source
4 = Laboratory – pathogen suspected to have caused illness identified in food handler
5 = Laboratory – pathogen suspected to have caused illness identified in implicated source (food)
6 = No evidence
7 = Other evidence

While a range of products were implicated as the suspected source of infection in the outbreaks, the level of confirmation for most outbreaks was low. In only one outbreak, linked to consumption of unpasteurised milk, was *Campylobacter* identified in the implicated source.

4.4.4.2 Laboratory investigation of samples from suspected foodborne outbreaks

During investigations of suspected foodborne illness outbreaks by ESR's Public Health Laboratory in 2009, *Campylobacter* was isolated from faecal samples from four investigations and one food sample (unpasteurised milk, see Northland outbreak in Table 12). The implicated foods in the four investigations with positive faecal samples were chicken (2), an Asian meal and unknown. For the investigation with unknown implicated food, both *Campylobacter* and norovirus were detected in a faecal sample.

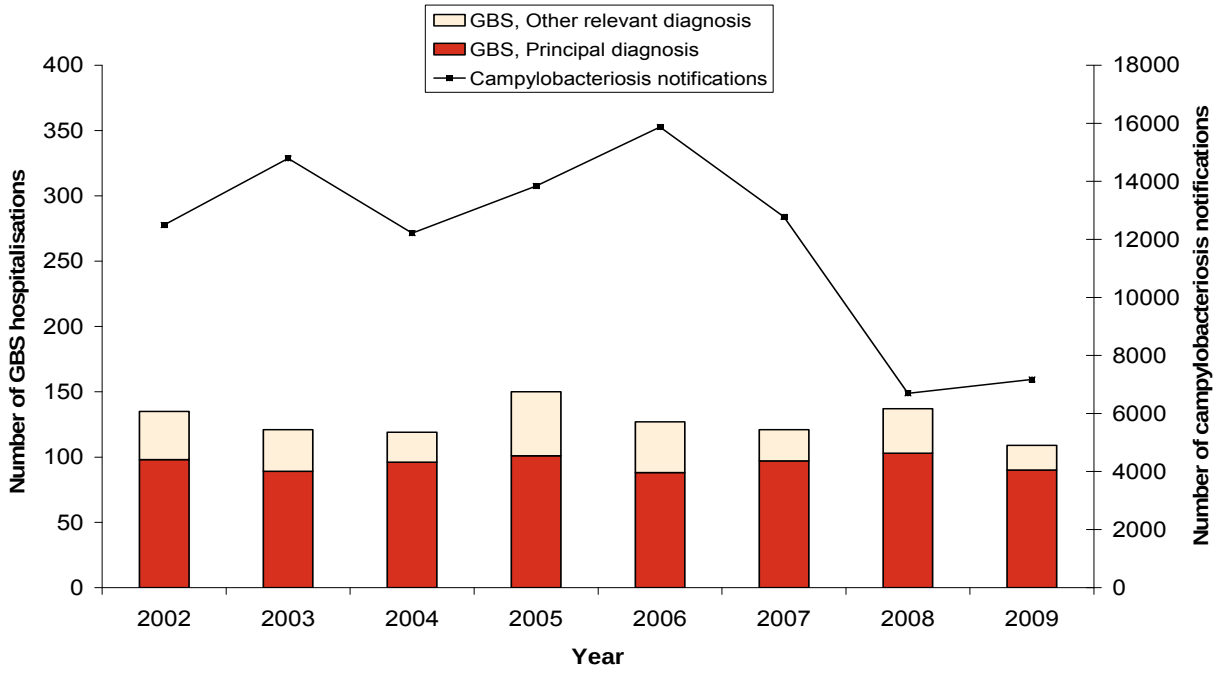
4.4.5 Disease sequelae - Guillain-Barré Syndrome (GBS)

Guillain-Barré Syndrome may be preceded by an infection with *Campylobacter jejuni*. Other respiratory or intestinal illnesses and other triggers may also precede an episode of GBS.

The ICD-10 code G61.0 was used to extract GBS hospitalisation data from the MoH NMDS database. Of the 109 hospitalised cases (2.5 admissions per 100 000 population) recorded in 2009, 90 were reported with GBS as the primary diagnosis and 19 with this condition as another relevant diagnosis.

Over the period 2002 to 2009, the number of hospitalised cases (any diagnosis code) for GBS have ranged from 109 to 150 (Figure 12). The numbers of campylobacteriosis notifications during the same period are also included in Figure 12, for comparison. There is little evidence for a correlation between campylobacteriosis notifications and hospitalised GBS cases, although the number of GBS cases in 2009 was the lowest reported during the period 2002-2009.

Figure 12: GBS hospitalised cases, 2002-2009



In 2009, the number of GBS hospital admissions was greater for males than females (Table 13).

Table 13: GBS hospitalised cases by sex, 2009

Sex	Cases hospitalised[a]	
	No.	Rate[b]
Male	64	3.0
Female	45	2.0
Total	109	2.5

[a] MoH morbidity data for hospital admissions
[b] per 100 000 of population

In 2009, the highest hospitalised case rate for GBS occurred in those aged 70+ years (Table 14).

Table 14: GBS hospitalised cases by age group, 2009

Age group	Cases hospitalised[a]	
	No.	Rate[b]
<5	4	-
5 to 9	3	-
10 to 14	7	2.4
15 to 19	4	-
20 to 29	12	2.1
30 to 39	11	1.9
40 to 49	16	2.5
50 to 59	15	2.8
60 to 69	15	3.8
70+	22	5.8
Total	109	1.3

[a] MoH morbidity data for hospital admissions
[b] per 100 000 of population. Where fewer than five cases have been reported a rate has not been calculated.

4.4.6 Relevant New Zealand studies and publications

4.4.6.1 Reports

In order to develop a more comprehensive picture of the contribution of *Campylobacter* from fresh retail poultry to human disease in New Zealand, and explore transmission cycles within and between these sources, studies of carriage of *Campylobacter* in end-of-lay meat breeders (also known as "spent hens"), ducks and turkeys were conducted (French, 2009). This is an extension of a three-year (2005-2008) project in the Manawatu aimed at source attribution of human campylobacteriosis cases using multi-locus sequence typing (MLST), which identified poultry as the most important source of infection (French, 2008). The 2009 study provides additional information on the carriage of *Campylobacter* spp. in non-broiler poultry sources in New Zealand, as well as an update on the recent trends of carriage in broiler chickens. *Campylobacter* spp. were present on most duck and turkey carcases examined, at similar concentrations to those found on broiler chickens. However, the genotypes of *C. jejuni* isolated from these sources were not commonly found in humans. Source attribution models therefore indicated a very low contribution to human infection from these sources. This may be due to these genotypes displaying lower pathogenicity but, given the relatively low consumption of these poultry sources, it is more likely that the low human case attribution merely reflects a lower exposure.

In a related study, campylobacteriosis notifications from 2001 to 2008 across three regions of New Zealand were analysed, and spatial and temporal trends were identified (Marshall *et al.*, 2009). Risk factors associated with these trends were investigated, and several relationships were observed. In urban areas, the Social Deprivation Index (SDI) was a risk factor for notifications, with areas of high deprivation having low notification rates. In contrast, the SDI had no clear association with notifications in rural areas, where areas of high ruminant (sheep and dairy) density were more closely aligned with notification rates. Differences were shown in notification rates across age groups, with children under 5 years of age having significantly higher notification rates than other age groups, with the majority of these notifications coming from rural populations. Multi-Locus Sequence Typing of isolates from the Manawatu indicate a clear difference in the spatial distribution of sequence types associated with poultry compared to those associated with ruminants, with poultry-associated isolates more prevalent in urban areas. Several meterological variables were also investigated, and were shown to be associated with the temporal variation in notification rates, though peaks in the weather variables lagged behind corresponding peaks in date of notification by several weeks.

Other studies reported during 2009 providing information on *Campylobacter* were:

- An investigation of rinsates classified as 'not detected' in the National Microbiological Databases (NMD) poultry *Campylobacter* programme, to determine whether a proportion of these rinsates were actually *Campylobacter*-positive, but with very low counts (Lake, 2009).
- An investigation of the reduction in *Campylobacter* on chicken breasts during commercial freezing and storage (McIntyre, 2009).
- A survey of microbiological hazards in conventional and organic fresh produce (McIntyre and Cornelius, 2009). *Campylobacter* was not detected in any of 891 samples of conventional (imported and domestic) or organic fresh fruits and vegetables.
- An investigation of the potential for farmers' overalls to be a transmission route for *Campylobacter* on broiler farms (Wong, 2009).

4.4.6.2 Journal papers

During 2009, two papers were published on the three-year (2005-2008) project in the Manawatu aimed at source attribution of human campylobacteriosis cases using multi-locus sequence typing (MLST) reported last year (French, 2008). One of the papers reported on the use of genetic and epidemiological modelling to determine campylobacteriosis source attribution (Mullner *et al.*, 2009b). Poultry was estimated to be the cause of 58-76% of human campylobacteriosis cases in New Zealand. The second paper was concerned with the use of a Bayesian modelling approach to source attribution (Mullner *et al.*, 2009a). The model assigned 80% of human campylobacteriosis cases to poultry sources, followed by bovine (10%), ovine (9%) and environmental sources (1%).

Several papers examined surface waters as a potential source of *Campylobacter*. Of 53 surface water samples from the Canterbury region, 45 (85%) were positive for *Campylobacter*, with concentrations in the range 0.4-110 MPN/100 ml (Bigwood and Hudson, 2009). Genetic analysis (MLST) of 244 *C. jejuni* isolates from three New Zealand river systems concluded that the majority of sequence types could be attributed to wild bird faecal contamination (Carter *et al.*, 2009). Two novel clonal complexes were identified, but the sequence types in these complexes have not been observed in human cases. No *Campylobacter* was isolated from 65 samples of watercress (*Nasturtium officinale),* collected from four streams in the Waitako region (Donnison *et al.*, 2009).

A review article compared risk assessments on *Campylobacter* in broiler meat from several countries, including New Zealand (Nauta *et al.*, 2009).

4.4.7 Relevant regulatory developments

NZFSA and the New Zealand Poultry Industry Association have developed a new code of practice (COP) specifically for poultry processing:
http://www.nzfsa.govt.nz/animalproducts/publications/code-of-practice/poultry/

The code includes:

- Improvements for control of *Campylobacter* identified by NZFSA's *Campylobacter* Strategy Working Group;
- Expected standards for Good Manufacturing Practice; and
- Procedures to promote compliance with legal requirements set under the Animal Products Act 1999

Further chapters of this Code were released or amended in 2009, including:

- Chapter 3: Hygiene and Sanitation;
- Chapter 5: Slaughter and Dressing; and
- Chapter 9: Secondary Processing.

4.5 Ciguatera Fish Poisoning (CFP)

4.5.1 Case definition

Clinical description:	Gastroenteritis, possibly followed by neurologic symptoms
Laboratory test for diagnosis:	Demonstration of ciguatoxin in implicated fish
Case classification:	Not applicable

4.5.2 Ciguatera fish poisoning cases reported in 2009 by data source

During 2009, eight notifications of ciguatera fish poisoning and no resulting deaths were reported in EpiSurv.

The ICD-10 code T61.0 was used to extract ciguatera fish poisoning hospitalisation data from the MoH NMDS database. Two hospital admissions were recorded in 2009, one with ciguatera fish poisoning as the primary diagnosis and other with ciguatera fish poisoning as another relevant diagnosis.

4.5.3 Outbreaks reported as caused by ciguatera fish poisoning

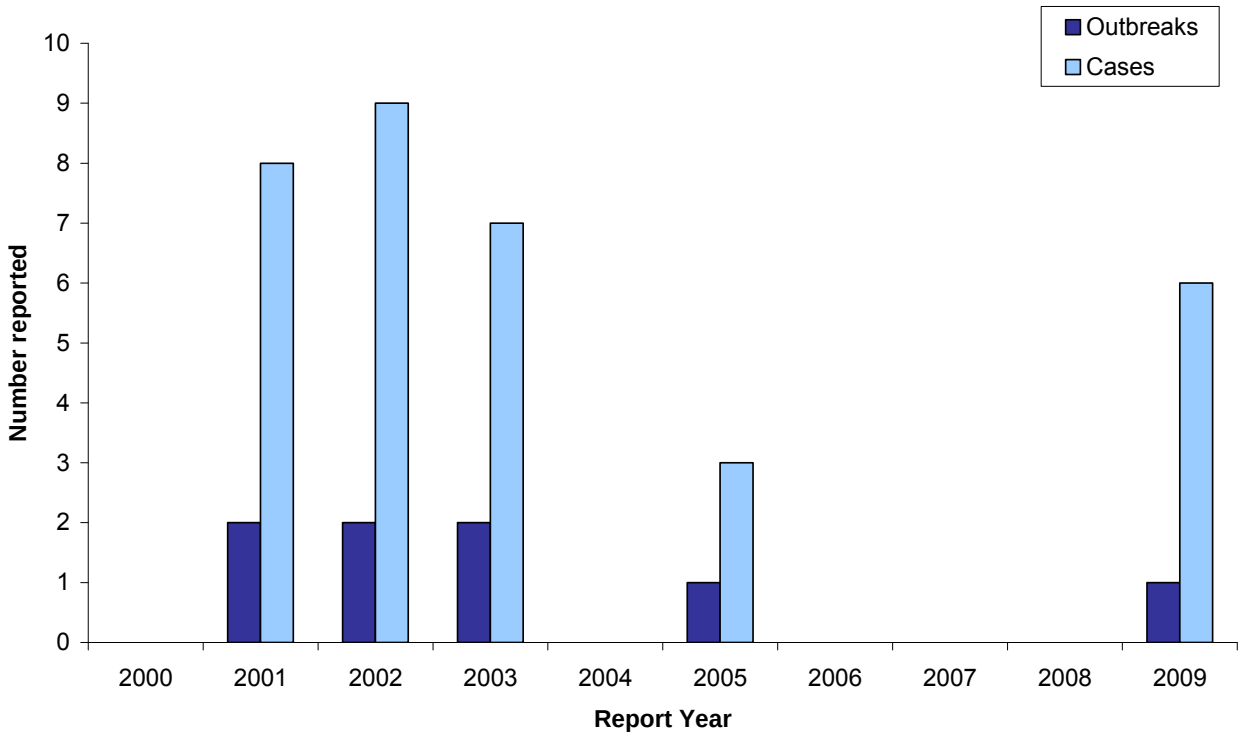
One foodborne ciguatera fish poisoning outbreak with six associated cases was reported in 2009 (Table 15).

Table 15: Details of food-associated ciguatera fish poisoning outbreak, 2009

Measure (No.)	Foodborne ciguatera fish poisoning outbreaks	All ciguatera fish poisoning outbreaks
Outbreaks	1	1
Cases	6	6
Hospitalised cases	0	0

Over the ten year period from 2000 to 2009, very few outbreaks of ciguatera fish poisoning have been reported, with no more than two outbreaks of ciguatera fish poisoning reported in any year (Figure 13).

Figure 13: Outbreaks and associated cases due to ciguatera fish poisoning reported by year, 2000-2009



4.5.3.1 Details of food-associated outbreaks

Table 16 contains details of the one food-associated ciguatera fish poisoning outbreak reported in 2009.

Table 16: Details of food-associated ciguatera fish poisoning outbreak

Public Health Unit (Month)	Suspected Vehicle	Setting	Number ill	Confirmation
Wellington (December)	Donu Fish	Home	6C	2, 5

C = confirmed, P = probable
Confirmation:
1 = Environmental investigation – identified critical control point failures linked to implicated source
2 = Epidemiological – case had history of exposure to implicated source
3 = Epidemiological – case control or cohort study showed elevated risk for cases to implicated source
4 = Laboratory – pathogen/chemical suspected to have caused illness identified in food handler
5 = Laboratory – pathogen/chemical suspected to have caused illness identified in implicated source (food)
6 = No evidence
7 = Other evidence

The single ciguatera fish poisoning outbreak reported included very strong evidence for Donu Fish as the source of the outbreak.

4.5.3.2 Laboratory investigation of samples from suspected foodborne outbreaks

Investigation of leftover Donu fish from the outbreak mentioned in the previous section resulted in detection of ciguatoxin by neuroblastoma assay. An associated faecal sample also contained high levels of *Clostridium perfringens* and *C. perfringens* enterotoxins.

4.5.4 Relevant New Zealand studies and publications

Nil.

4.5.5 Relevant regulatory developments

A recall of warm water whole frozen fish species, purchased from specified retail outlets in the Wellington region was initiated during December 2009, due to risks of ciguatera fish poisoning[2]

4.6 *Clostridium perfringens* Intoxication

4.6.1 Case definition

Clinical description: Gastroenteritis with profuse watery diarrhoea

Laboratory test for diagnosis: Detection of enterotoxin in faecal specimen or faecal spore count of $\geq 10^6$/g or isolation of $\geq 10^5$/g *C. perfringens* in leftover food

Case classification:

Probable: A clinically compatible illness that is either a contact of a confirmed case of the same disease, or has had contact with the same common source i.e., is part of an identified common source outbreak

Confirmed: A clinically compatible illness that is laboratory confirmed

[2] http://www.nzfsa.govt.nz/processed-food-retail-sale/recalls/products/2009/recalled-food-products-cigtoxic-fish.htm

4.6.2 *Clostridium perfringens* intoxication cases reported in 2009 by data source

During 2009, one notification of *Clostridium perfringens* intoxication and no resulting deaths were reported in EpiSurv.

The ICD-10 code A05.2 was used to extract foodborne *Clostridium perfringens* intoxication hospitalisation data from the MoH NMDS database. There were no hospital admissions recorded in 2009 with *Clostridium perfringens* intoxication as a primary or other relevant diagnosis.

4.6.3 Outbreaks reported as caused by *Clostridium perfringens*

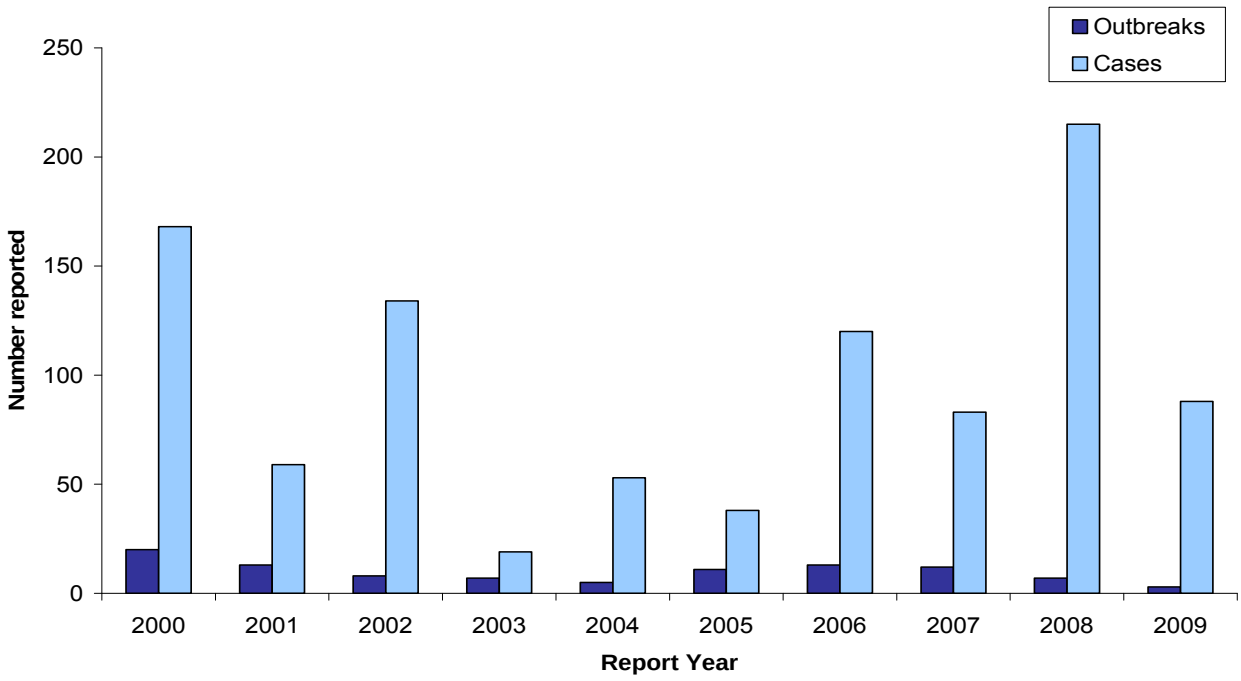
All three *Clostridium perfringens* outbreaks for 2009 were associated with a suspected or known foodborne source (Table 17).

Table 17: *Clostridium perfringens* outbreaks reported, 2009

Measure (No.)	Foodborne *Clostridium perfringens* outbreaks	All *Clostridium perfringens* outbreaks
Outbreaks	3	3
Cases	88	88
Hospitalised cases	0	0

Since 2000, the number of foodborne outbreaks associated with *Clostridium perfringens* has fluctuated, from three in 2009 to 20 outbreaks in 2000 (Figure 14). The number of cases associated with *Clostridium perfringens* outbreaks has also varied over time. In 2008, the number of cases (215) associated with foodborne outbreaks due to *Clostridium perfringens* was the highest of any year in the period monitored (2000-2009).

Figure 14: Foodborne *Clostridium perfringens* outbreaks and associated cases reported by year, 2000-2009



4.6.3.1 Details of food-associated outbreaks

Table 18 contains details of the three food–associated *Clostridium perfringens* outbreaks reported in 2009.

Table 18: Details of food-associated *Clostridium perfringens* outbreaks, 2009

Public Health Unit (Month)	Suspected vehicle	Setting	Number ill	Confirmation
Auckland (July)	Fish and chips	Restaurant/Café	2P	1, 2
Nelson (April)	Chicken and rice	Workplace	4C, 74P	1, 2, 5
Wellington (November)	Unknown	Hotel/Motel	8C	7

C = confirmed, P = probable
Confirmation:
1 = Environmental investigation – identified critical control point failures linked to implicated source
2 = Epidemiological – case had history of exposure to implicated source
3 = Epidemiological – case control or cohort study showed elevated risk for cases to implicated source
4 = Laboratory – pathogen suspected to have caused illness identified in food handler
5 = Laboratory – pathogen suspected to have caused illness identified in implicated source (food)
6 = No evidence
7 = Other evidence

Evidence linking *Clostridium perfringens* outbreaks to particular food vehicles was weak for two of the three outbreaks. However, the largest outbreak occurring in April included very strong evidence for a chicken and rice meal as the source of the outbreak.

4.6.3.2 Laboratory investigation of samples from suspected foodborne outbreaks

During investigations of suspected foodborne illness outbreaks by ESR's Public Health Laboratory, *Clostridium perfringens* and/or its toxin was detected in clinical samples from five investigations, while high levels of *Clostridium perfringens* were detected in the associated food from one of these investigations (butter chicken and rice). Implicated foods from a further two of these investigations were fish and chips and roast chicken. No specific foods were implicated in the remaining two investigations.

4.6.4 Relevant New Zealand studies and publications

Nil.

4.6.5 Relevant regulatory developments

Nil.

4.7 Cryptosporidiosis

Summary data for cryptosporidiosis in 2009 are given in Table 19.

Table 19: Summary surveillance data for cryptosporidiosis, 2009

Parameter	Value in 2009	Section reference
Number of cases	854	4.7.2
Rate (per 100 000)	19.8	4.7.2
Hospitalisations (%)	23 (2.7%)	4.7.2
Deaths (%)	0 (0%)	4.7.2
Estimated travel-related cases (%)	74 (8.6%)	4.7.3.6
Estimated food-related cases (%)	NA	

NA = not applicable, no information is available on the food attributable proportion of cryptosporidiosis in New Zealand

4.7.1 Case definition

Clinical description: An illness with diarrhoea and abdominal pain. The infection may be asymptomatic

Laboratory test for diagnosis: Detection of *Cryptosporidium parvum* oocysts in a faecal specimen

Case classification:

Probable A clinically compatible illness that is either a contact of a confirmed case of the same disease, or has had contact with the same common source i.e., is part of an identified common source outbreak

Confirmed A clinically compatible illness that is laboratory confirmed

4.7.2 Cryptosporidiosis cases reported in 2009 by data source

During 2009, 854 notifications (19.8 cases per 100 000 population) of cryptosporidiosis and no resulting deaths were reported in EpiSurv.

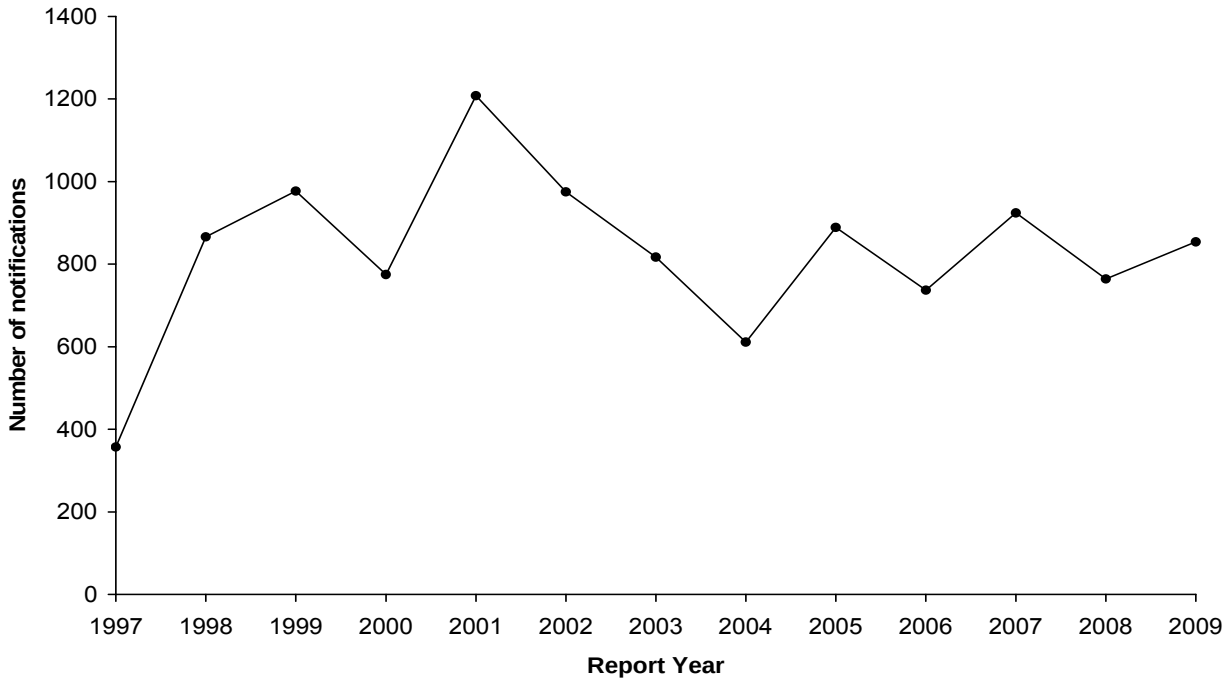
The ICD-10 code A07.2 was used to extract cryptosporidiosis hospitalisation data from the MoH NMDS database. Of the 23 hospital admissions (0.5 admissions per 100 000 population) recorded in 2009, 19 were reported with cryptosporidiosis as the primary diagnosis and four with cryptosporidiosis as another relevant diagnosis.

4.7.3 Notifiable disease data

4.7.3.1 Annual notification trend

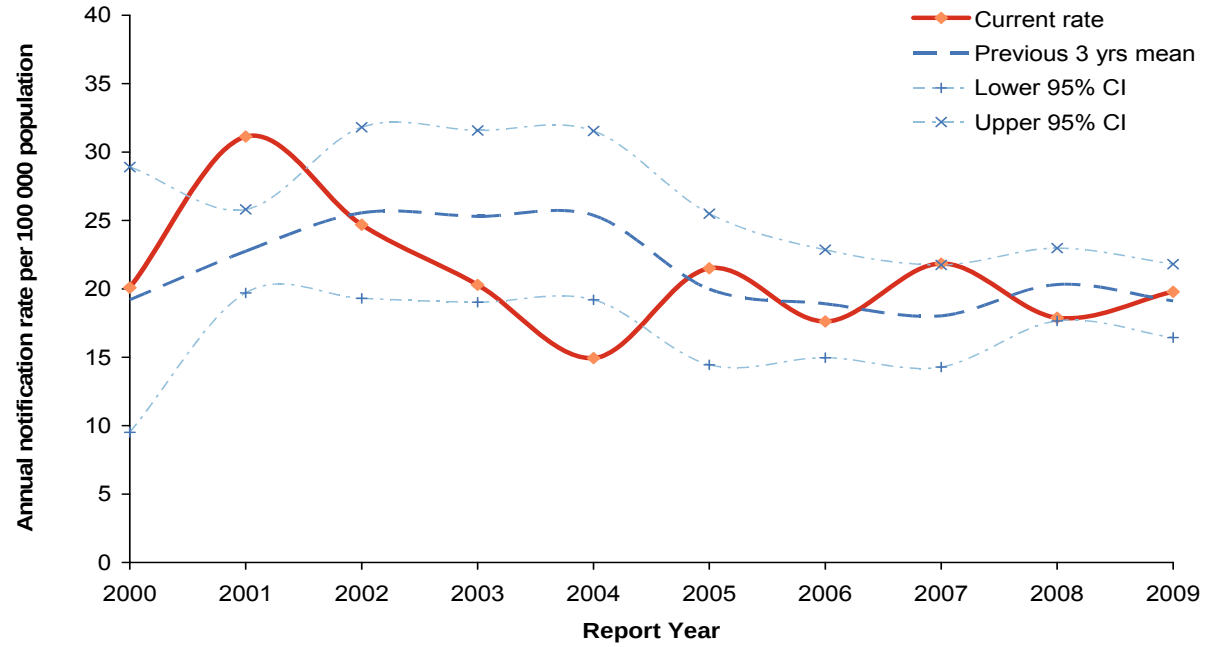
Cryptosporidiosis became a notifiable disease in 1996. The number of notifications peaked at 1 208 cases in 2001 and then decreased to 611 in 2004. Since 2004 the number of notifications has fluctuated between 737 (2006) and 924 (2007) (Figure 15).

Figure 15: Cryptosporidiosis notifications by year, 1997-2009



The cryptosporidiosis annual population rate trend is very similar to the corresponding annual notification trend. The highest cryptosporidiosis annual notification rate was reported in 2001 and generally decreased until 2004. Notification rates have fluctuated since 2004, but generally slightly higher rates have been observed than in 2004 (Figure 16).

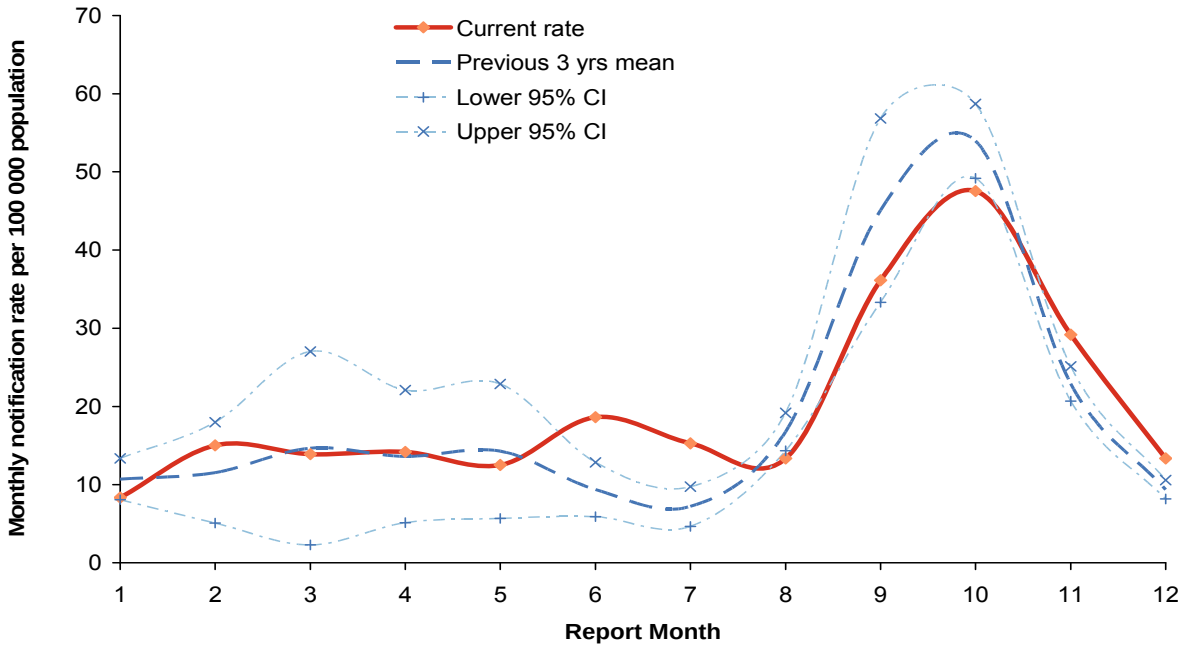
Figure 16: Cryptosporidiosis notification rate by year, 2000-2009



4.7.3.2 Seasonality

The number of notified cases of cryptosporidiosis reported per 100 000 population by month for 2009 was similar to previous years. Cryptosporidiosis has a consistent spring peak that occurs each year in September or October (Figure 17).

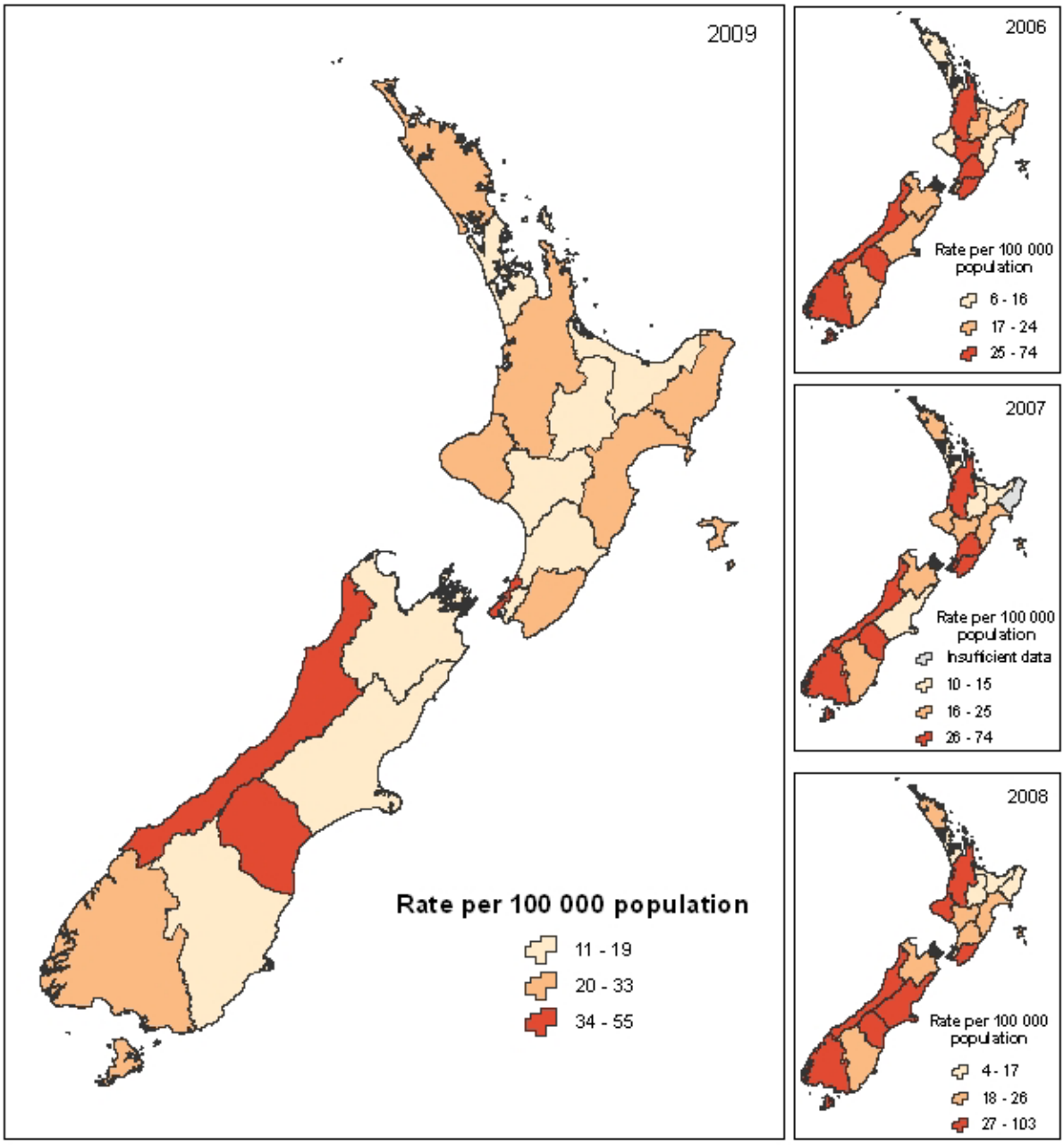
Figure 17: Cryptosporidiosis monthly rate (annualised) for 2009



4.7.3.3 Geographic distribution of cryptosporidiosis notifications

There have been consistently higher population rates of cryptosporidiosis notifications in the predominantly rural DHBs compared to the more urban DHBs (Figure 18). In 2009, the highest rates were reported in West Coast (55.2 per 100 000 population, 18 cases) and South Canterbury (50.4 per 100 000, 28 cases) DHBs. The lowest rate was reported in Counties Manukau DHB (10.6 per 100 000, 51 cases). West Coast and South Canterbury DHBs have been in the highest quantile of cryptosporidiosis notification rates for each of the last four years.

Figure 18: Geographic distribution of cryptosporidiosis notifications, 2006-2009



4.7.3.4 Age and sex distribution of cryptosporidiosis cases

In 2009, the number and notification rates for cryptosporidiosis were slightly higher for females compared to males. However the number of hospitalisations was similar for females and males (Table 20).

Table 20: Cryptosporidiosis cases by sex, 2009

Sex	EpiSurv notifications		Morbidity[a]		Deaths recorded in EpiSurv
	No.	Rate[b]	No.	Rate[b]	No.
Male	384	18.1	13	0.6	
Female	463	21.1	10	0.5	
Unknown	7				
Total	854	19.8	23	0.5	

[a] MoH morbidity data for hospital admissions
[b] per 100 000 of population

During 2009, the highest cryptosporidiosis age specific notification rates were in the 1 to 4 years age group (110.1 per 100 000 population, 267 cases), followed by the less than one year age group (46.0 per 100 000, 29 cases) and the 5 to 9 years age group (35.4 per 100 000, 102 cases) (Table 21). The hospitalisation rate was not defined for most age groups due to the small number of cases.

Table 21: Cryptosporidiosis cases by age group, 2009

Age group	EpiSurv notifications		Morbidity[a]		Deaths recorded in EpiSurv
	No.	Rate[b]	No.	Rate[b]	No.
<1	29	46.0	2	-	
1 to 4	267	110.1	1	-	
5 to 9	102	35.4	0	-	
10 to 14	69	23.2	5	1.7	
15 to 19	44	13.6	5	1.5	
20 to 29	93	15.9	4	-	
30 to 39	131	22.7	3	-	
40 to 49	58	9.1	0	-	
50 to 59	30	5.6	1	-	
60 to 69	15	3.8	0	-	
70+	13	3.4	2	-	
Unknown	3				
Total	854	19.8	23	0.5	

[a] MoH morbidity data for hospital admissions
[b] per 100 000 of population. Where fewer than five cases have been reported a rate has not been calculated.

4.7.3.5 Risk Factors Reported

During 2009, the most commonly reported risk factors reported for cryptosporidiosis were contact with farm animals (55.1%), contact with faecal matter (33.7%), and consumption of untreated water (31.7%) (Table 22).

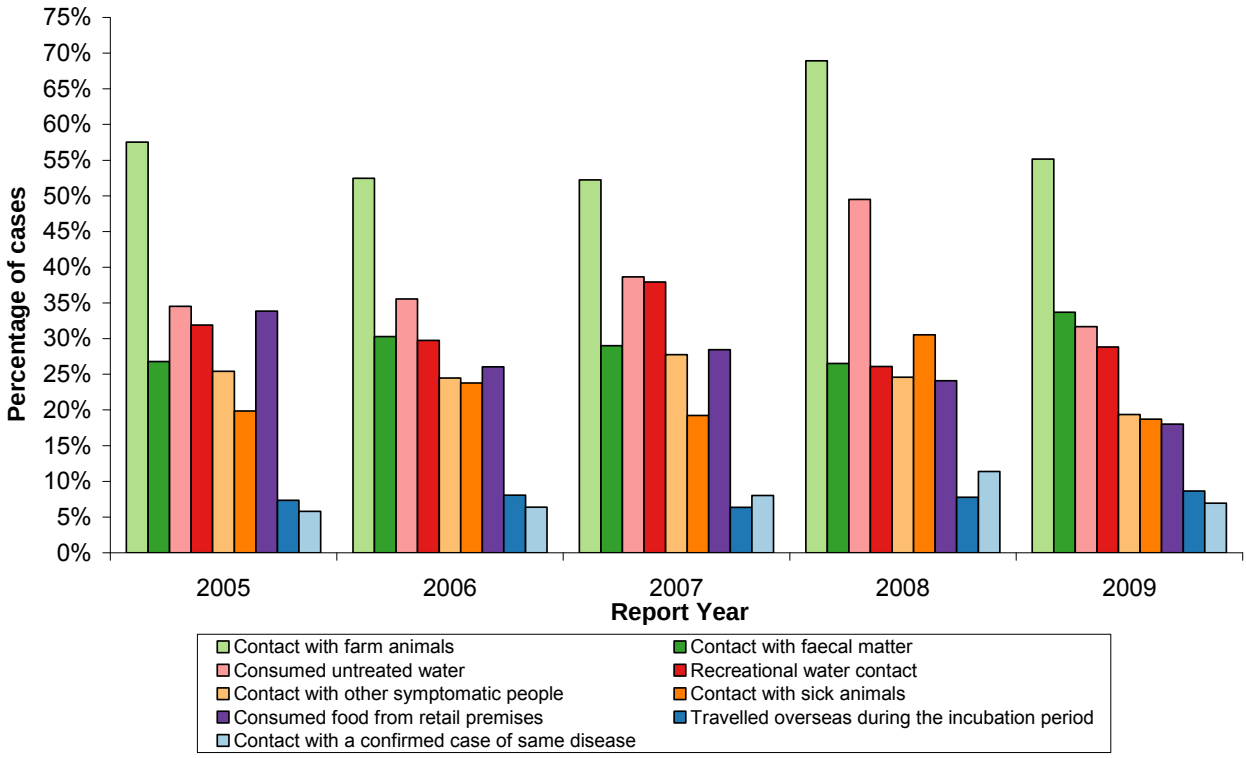
Table 22: Exposure to risk factors associated with cryptosporidiosis, 2009

	Notifications			
Risk Factor	**Yes**	**No**	**Unknown**	**%[a]**
Contact with farm animals	241	196	417	55.1
Contact with faecal matter	123	242	489	33.7
Consumed untreated water	108	233	513	31.7
Recreational water contact	124	306	424	28.8
Contact with other symptomatic people	75	312	467	19.4
Contact with sick animals	70	304	480	18.7
Consumed food from retail premises	66	300	488	18.0
Travelled overseas during the incubation period	40	423	391	8.6
Contact with a confirmed case of same disease	24	321	509	7.0

[a]Percentage refers to the cases that answered "yes" out of the total number of cases for which this information was supplied. Cases may have more than one risk factor recorded.

Between 2005 and 2009, the most consistently reported risk factors for cryptosporidiosis were contact with farm animals and consumption of untreated water (Figure 19).

Figure 19: Cryptosporidiosis risk factors by percentage of cases and year, 2005-2009



4.7.3.6 Estimate of travel-related cases

For cases where information on travel was provided, 8.6% (95%CI 6.2-11.6%) had travelled overseas during the incubation period. Assuming that the cases for which travel information was provided were representative of all cryptosporidiosis cases, a Poisson distribution can be used to estimate the total number of potentially travel related cases of cryptosporidiosis in 2009. The resultant distribution has a mean of 74 cases (95% CI 51-99).

4.7.4 Outbreaks reported as caused by *Cryptosporidium* spp.

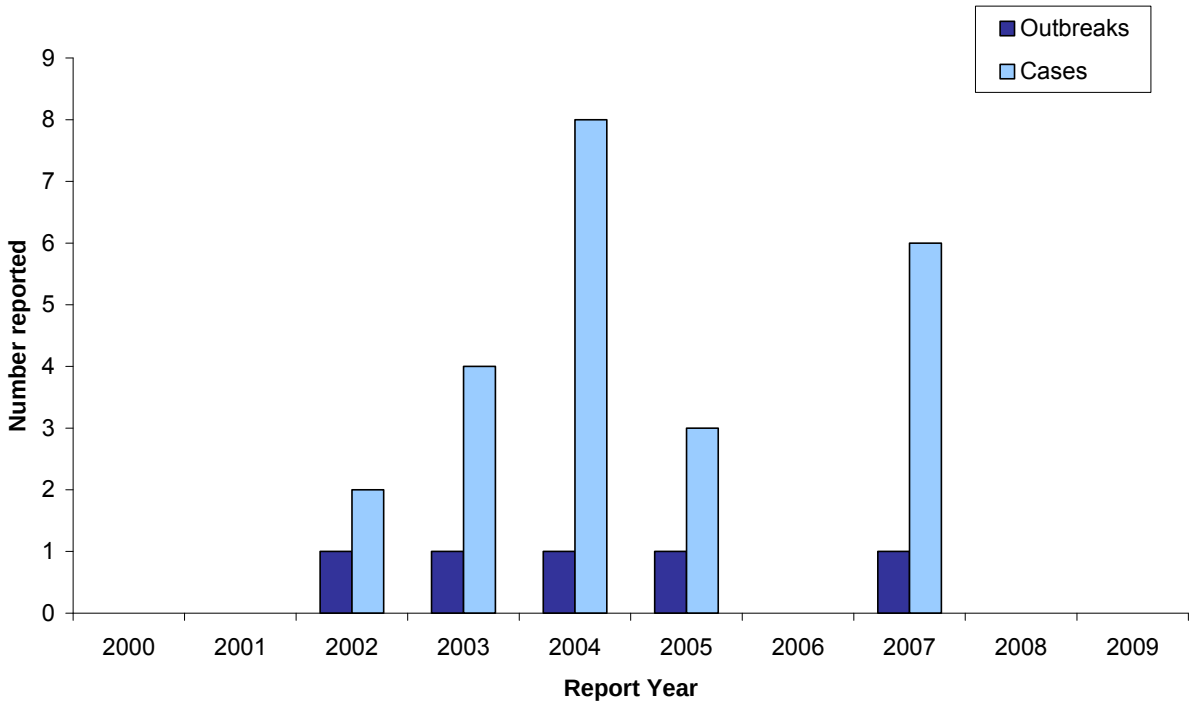
No foodborne *Cryptosporidium* outbreaks were reported in 2009 (Table 23).

Table 23: *Cryptosporidium* spp. outbreaks reported, 2009

Measure (No.)	Foodborne *Cryptosporidium* spp. outbreaks	All *Cryptosporidium* spp. outbreaks
Outbreaks	0	20
Cases	0	68
Hospitalised cases	0	0

Foodborne *Cryptosporidium* outbreaks are rare with not more than one outbreak reported each year in the ten year period, 2000-2009 (Figure 20). The largest outbreak, with eight associated cases, was reported in 2004.

Figure 20: Foodborne *Cryptosporidium* spp. outbreaks and associated cases reported by year, 2000–2009



4.7.4.1 Details of food-associated outbreaks

No foodborne *Cryptosporidium* outbreaks were reported in 2009.

4.7.4.2 Laboratory investigation of samples from suspected foodborne outbreaks

During investigations of suspected foodborne illness outbreaks by ESR's Public Health Laboratory, *Cryptosporidium* spp. was detected in two samples, one of human faeces and one of animal (cow) faeces. However, neither investigation implicated food as the source of the infection.

4.7.5 Relevant New Zealand studies and publications

4.7.5.1 Journal papers

A description of the epidemiology of cryptosporidiosis in New Zealand found a correlation between local notification rates and farm animal density, and concluded that transmission was mostly from farm animals to humans (Snel *et al.*, 2009a; Snel *et al.*, 2009b).

4.7.6 Relevant regulatory developments

Nil.

4.8 Giardiasis

Summary data for giardiasis in 2009 are given in Table 24.

Table 24: Summary surveillance data for giardiasis, 2009

Parameter	Value in 2009	Section reference
Number of cases	1 640	4.8.2
Rate (per 100 000)	38.0	4.8.2
Hospitalisations (%)	34 (2.7%)	4.8.2
Deaths (%)	0 (0%)	4.8.2
Estimated travel-related cases (%)	295 (18.0%)	4.8.3.6
Estimated food-related cases (%)	NA	

NA = not applicable, no information is available on the food attributable proportion of giardiasis in New Zealand

4.8.1 Case definition

Clinical description: An illness characterised by diarrhoea, abdominal cramps, bloating, weight loss or malabsorption. The infection may be asymptomatic

Laboratory test for diagnosis: Detection of *Giardia* cysts or trophozoites in a specimen from the human intestinal tract OR detection of *Giardia* antigen in faeces

Case classification:

Probable A clinically compatible illness that is either a contact of a confirmed case of the same disease, or has had contact with

	the same common source i.e., is part of an identified common source outbreak
Confirmed	A clinically compatible illness that is laboratory confirmed

4.8.2 Giardiasis cases reported in 2009 by data source

During 2009, 1 640 notifications (38.0 cases per 100 000 population) of giardiasis and no resulting deaths were reported in EpiSurv.

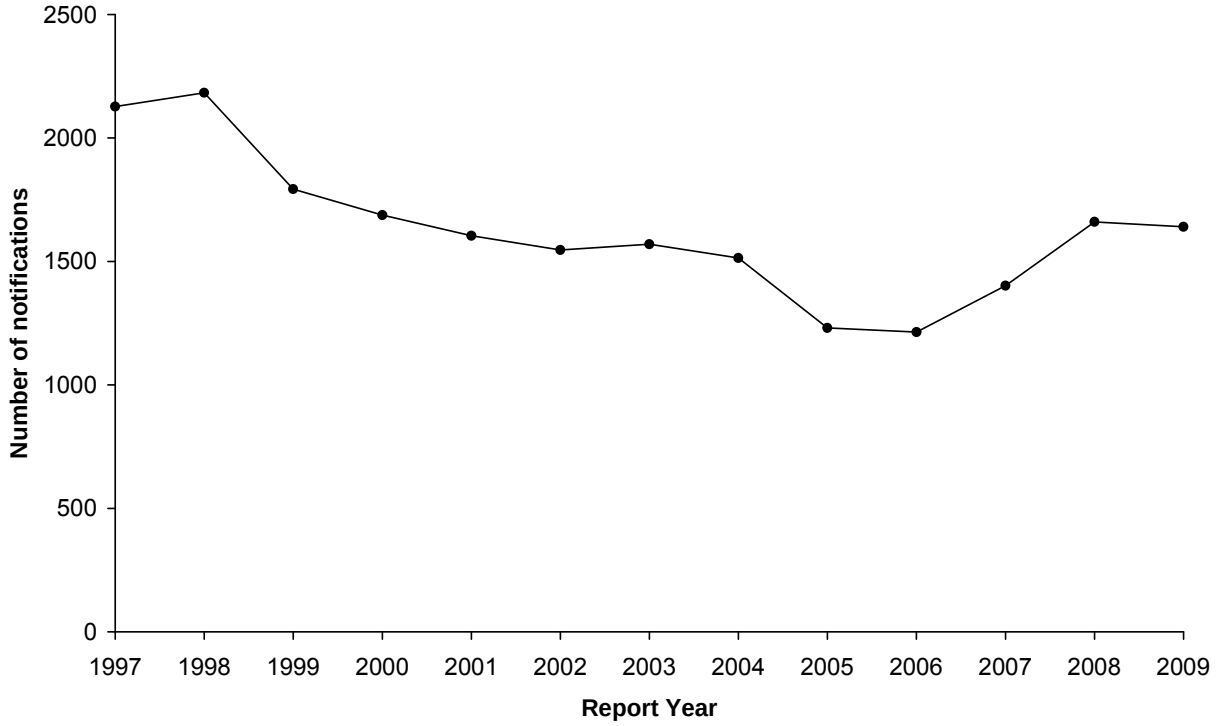
The ICD-10 code A07.1 was used to extract giardiasis hospitalisation data from the MoH NMDS database. Of the 34 hospital admissions (0.8 admissions per 100 000 population) recorded in 2009, 21 were reported with giardiasis as the primary diagnosis and 13 with giardiasis as another relevant diagnosis.

4.8.3 Notifiable Disease Data

4.8.3.1 Annual notification trend

Giardiasis became a notifiable disease in 1996. From 1998, there was a steady decrease in the number of cases reported each year up until 2006. Recent years have seen an increase in notifications, although the number of cases in 2009 was very similar to the number in 2008 (Figure 21).

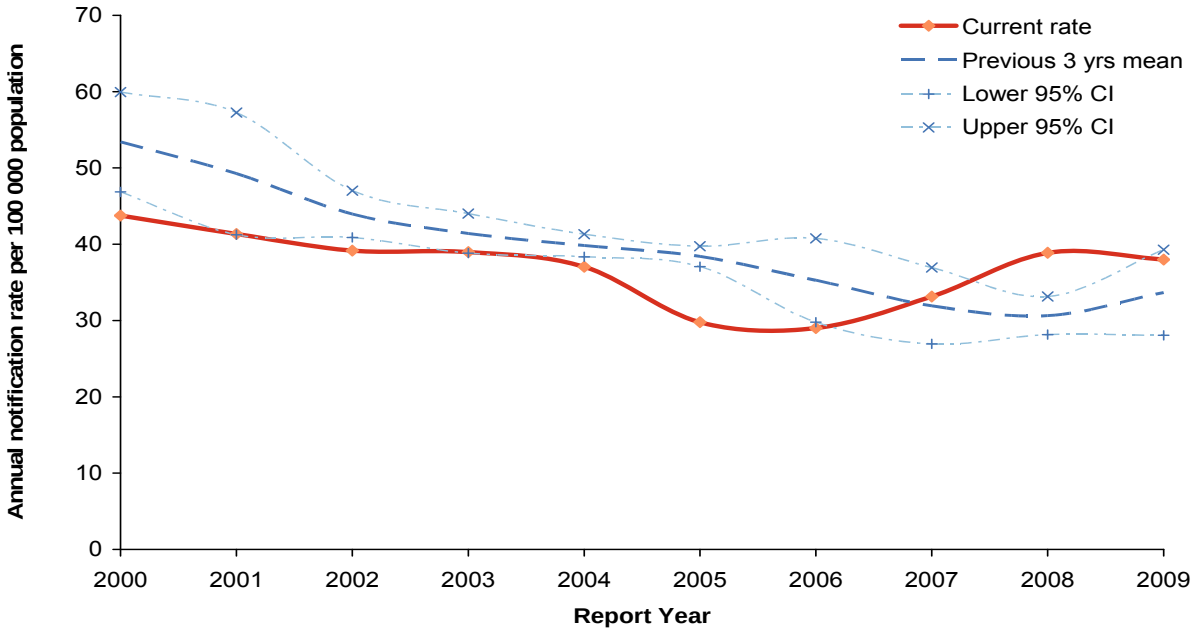
Figure 21: Giardiasis notifications by year, 1996-2009



The giardiasis annual population rate trend is very similar to the corresponding annual notification trend. The giardiasis notification rate had steadily declined from 43.8 per 100 000 population in

2000 to 29.0 per 100 000 in 2006, but increased steadily from 2006 to 2008 (Figure 22). The rate in 2009 was very similar to the rate in 2008.

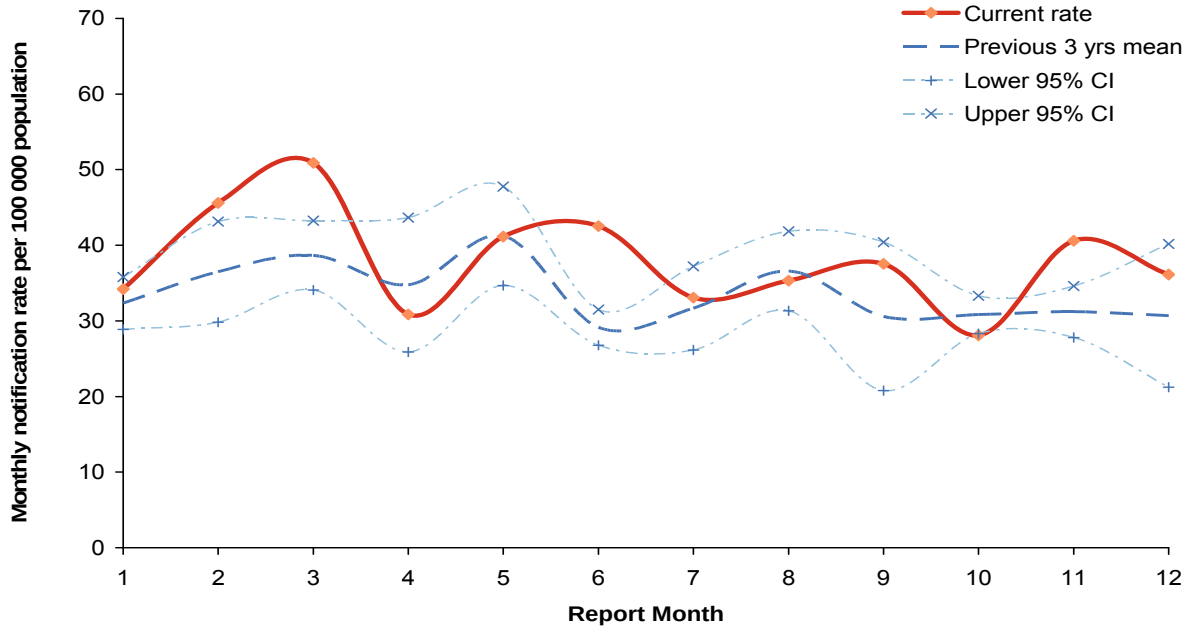
Figure 22: Giardiasis notification rate by year, 2000-2009



4.8.3.2 Seasonality

There was no strong seasonal pattern in the population rate of giardiasis notifications reported by month either historically or in 2009 (Figure 23).

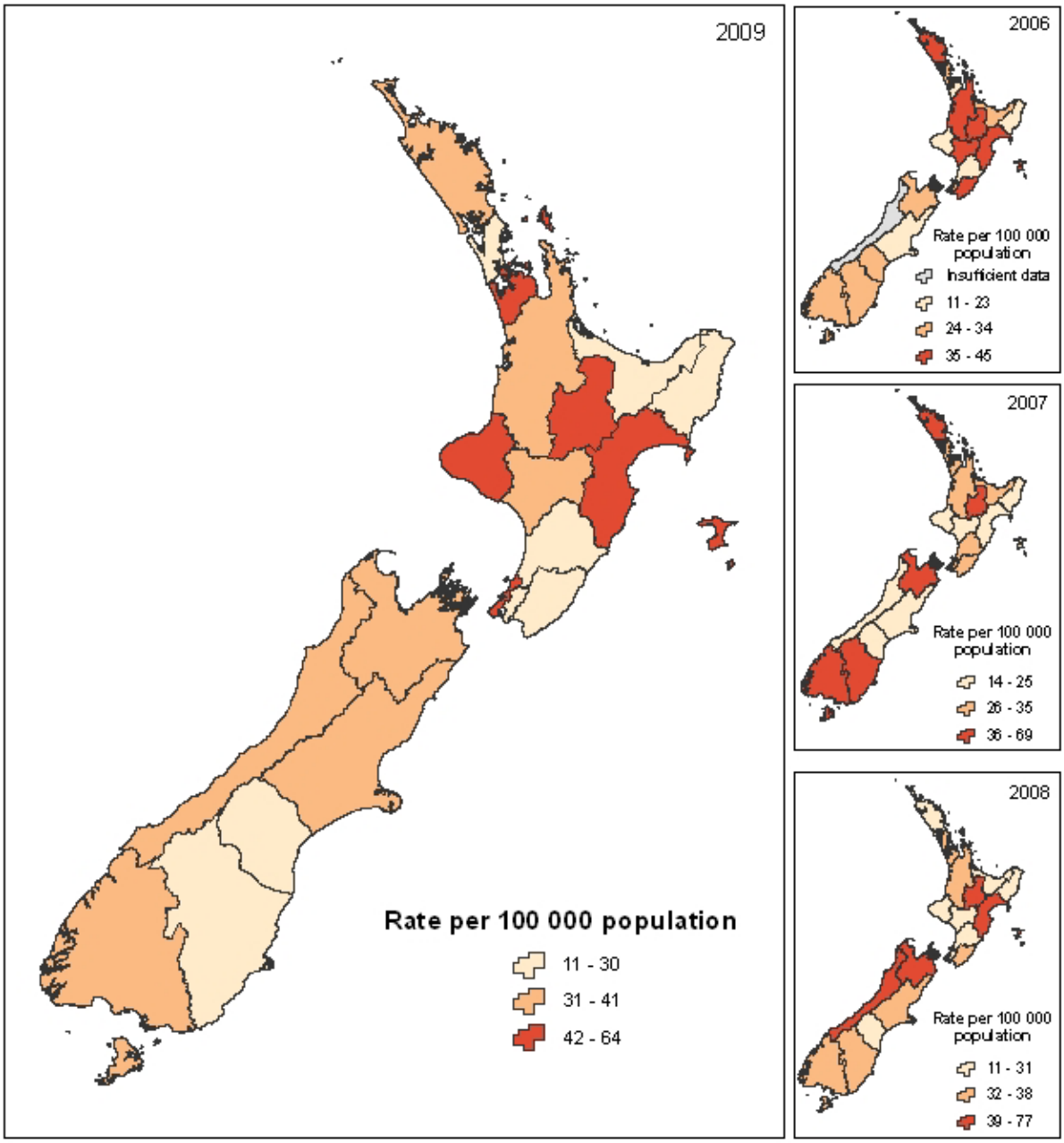
Figure 23: Giardiasis monthly rate (annualised) for 2009



4.8.3.3 Geographic distribution of giardiasis notifications

Notification rates of giardiasis varied throughout the country during 2009 (Figure 24). The highest rates were recorded in Lakes DHB (63.9 per 100 000 population, 65 cases), followed by Capital and Coast (58.0 per 100 000, 167 cases) and Auckland (47.7 per 100 000, 212 cases) DHBs. The lowest rate was recorded in MidCentral DHB (10.8 per 100 000, 18 cases). Lakes DHB has been consistently in the highest quantile of giardiasis notification rates for each of the last four years.

Figure 24: Geographic distribution of giardiasis notifications, 2006-2009



4.8.3.4 Age and sex distribution of giardiasis cases

The 2009 giardiasis notification and hospitalisation rates were higher for males compared to females (Table 25).

Table 25: Giardiasis cases by sex, 2009

Sex	EpiSurv notifications		Morbidity[a]		Deaths recorded in EpiSurv
	No.	Rate[b]	No.	Rate[b]	No.
Male	838	39.6	21	1.0	
Female	781	35.5	13	0.6	
Unknown	21				
Total	1 640	38.0	34	0.8	

[a] MoH morbidity data for hospital admissions
[b] per 100 000 of population

In 2009, the highest age-specific giardiasis notification rates were in those aged one to four years (136.5 per 100 000 population, 331 cases) followed by the 30 to 39 years age group (64.7 per 100 000, 373 cases) and the less than one year age group (57.1 per 100 000, 36 cases) (Table 26). The hospitalisation rate was not defined for most age groups due to the small number of cases.

Table 26: Giardiasis cases by age group, 2009

Age group	EpiSurv notifications		Morbidity[a]		Deaths recorded in EpiSurv
	No.	Rate[b]	No.	Rate[b]	No.
<1	36	57.1	3	-	
1 to 4	331	136.5	2	-	
5 to 9	127	44.1	1	-	
10 to 14	55	18.5	2	-	
15 to 19	23	7.1	0	-	
20 to 29	153	26.2	5	0.9	
30 to 39	373	64.7	9	1.6	
40 to 49	243	38.3	0	-	
50 to 59	153	28.8	5	0.9	
60 to 69	111	28.3	4	-	
70+	26	6.8	3	-	
Unknown	9				
Total	1 640	38.0	34	0.8	

[a] MoH morbidity data for hospital admissions
[b] per 100 000 of population. Where fewer than five cases have been reported a rate has not been calculated.

4.8.3.5 Risk Factors Reported

In 2009, the most commonly reported risk factors for notified giardiasis cases were consumption of untreated water (36.9%), contact with other symptomatic people (36.6%), and contact with faecal matter (35.3%) (Table 27).

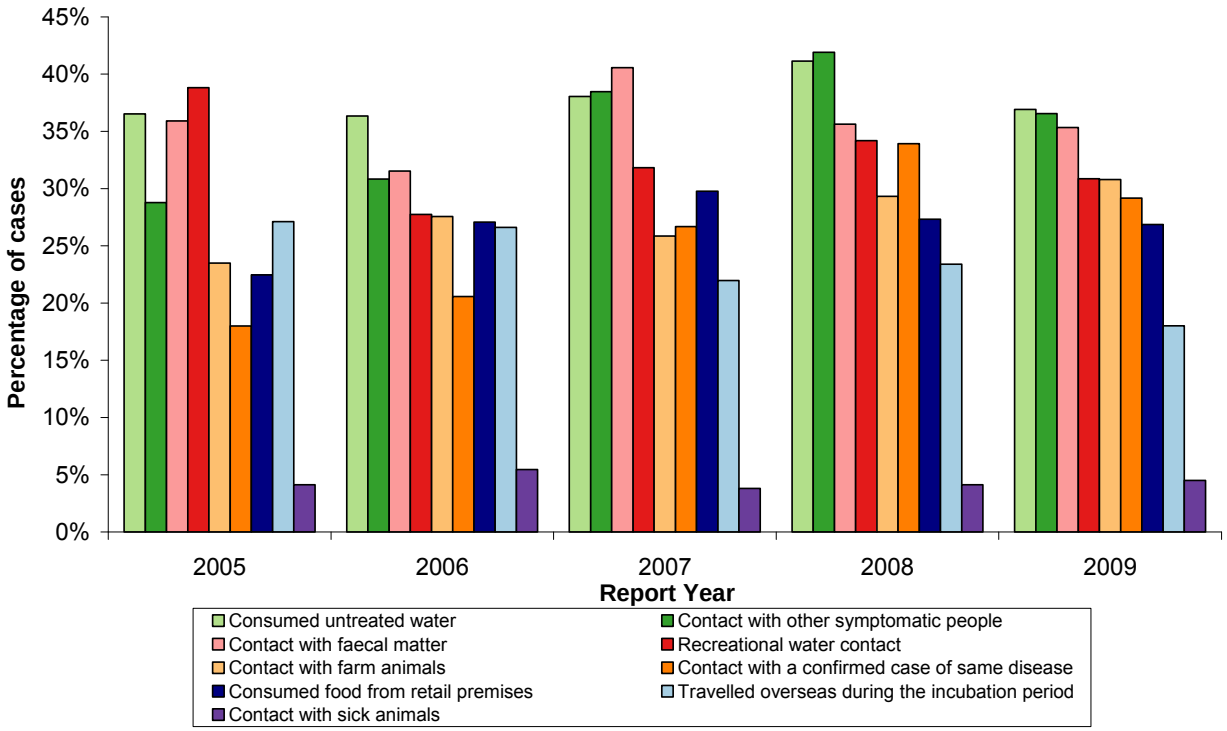
Table 27: Exposure to risk factors associated with giardiasis, 2009

	Notifications			
Risk Factor	**Yes**	**No**	**Unknown**	**%[a]**
Consumed untreated water	172	294	1 174	36.9
Contact with other symptomatic people	204	354	1 082	36.6
Contact with faecal matter	171	313	1 156	35.3
Recreational water contact	163	365	1 112	30.9
Contact with farm animals	177	398	1 065	30.8
Contact with a confirmed case of same disease	154	374	1 112	29.2
Consumed food from retail premises	127	346	1 167	26.8
Travelled overseas during the incubation period	114	519	1 007	18.0
Contact with sick animals	24	508	1 108	4.5

[1]Percentage refers to the cases that answered "yes" out of the total number of cases for which this information was supplied. Cases may have more than one risk factor recorded.

Between 2005 and 2009, the most consistently reported risk factors for giardiasis were consumption of untreated water, contact with faecal matter and contact with other symptomatic people (Figure 25).

Figure 25: Giardiasis risk factors by percentage of cases and year, 2005-2009



4.8.3.6 Estimate of travel-related cases

For cases where information on travel was provided, 18.0% (95%CI 15.1-21.2%) had travelled overseas during the incubation period. Assuming that the cases for which travel information was provided were representative of all giardiasis cases, a Poisson distribution can be used to estimate the total number of potentially travel related cases of giardiasis in 2009. The resultant distribution has a mean of 295 cases (95% CI 244-351).

4.8.4 Outbreaks reported as caused by *Giardia* spp.

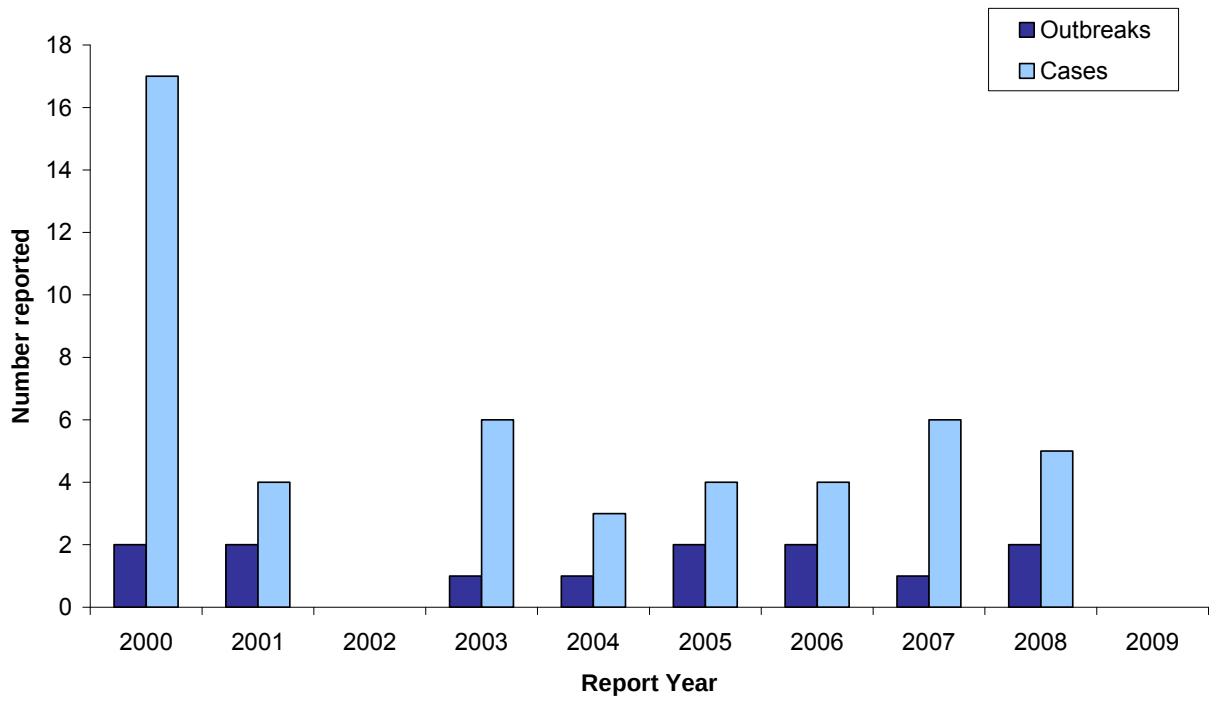
In 2009, there were 41 *Giardia* spp. outbreaks reported. However, none of these was associated with a suspected or known foodborne source (Table 28).

Table 28: *Giardia* spp. outbreaks reported, 2009

Measure (No.)	Foodborne *Giardia* spp. outbreaks	All *Giardia* spp. outbreaks
Outbreaks	0	41
Cases	0	131
Hospitalised cases	0	0

Since 2000, one or two foodborne *Giardia* spp. outbreaks have been reported in EpiSurv each year, with the exception of 2002 and 2009 where no outbreaks were reported (Figure 26). These outbreaks involved small numbers of cases.

Figure 26: Foodborne *Giardia* outbreaks and associated cases of reported by year, 2000-2009



4.8.4.1 Details of food-associated outbreaks

No foodborne *Giardia* spp. outbreaks were reported in 2009.

4.8.4.2 Laboratory investigation of samples from suspected foodborne outbreaks

During investigations of suspected foodborne illness outbreaks by ESR's Public Health Laboratory, no samples were found to contain *Giardia* spp.

4.8.5 Relevant New Zealand studies and publications

4.8.5.1 Journal papers

A description of the epidemiology of giardiasis in New Zealand concluded that the distribution of cases was consistent with largely human reservoirs, with a relatively small contribution from zoonotic sources in rural environments and a modest contribution from overseas travel (Snel *et al.*, 2009a; Snel *et al.*, 2009b).

4.8.6 Relevant regulatory developments

Nil.

4.9 Hepatitis A

Summary data for hepatitis A in 2009 are given in Table 29.

Table 29: Summary surveillance data for hepatitis A, 2009

Parameter	Value in 2009	Section reference
Number of cases	44	4.9.2
Rate (per 100,000)	1.0	4.9.2
Hospitalisations (%)	24 (54.5%)	4.9.2
Deaths (%)	0 (0%)	4.9.2
Estimated travel-related cases (%)	33 (75.0%)	4.9.3.6
Estimated food-related cases (%)	NA	

NA = not applicable, no information is available on the food attributable proportion of hepatitis A in New Zealand

4.9.1 Case definition

Clinical description: An illness with a discrete onset of symptoms (fever, malaise, anorexia, nausea, or abdominal discomfort) with jaundice and/or elevated serum aminotransferase levels

Laboratory test for diagnosis: Positive anti HAV IgM in serum

Case classification:

Probable: A clinically compatible illness that is either a contact of a confirmed case of the same disease, or has had contact with the same common source i.e., is part of an identified common source outbreak

Confirmed A clinically compatible illness that is laboratory confirmed

4.9.2 Hepatitis A cases reported in 2009 by data source

During 2009, 44 notifications (1.0 cases per 100 000 population) of hepatitis A and no resulting deaths were reported in EpiSurv.

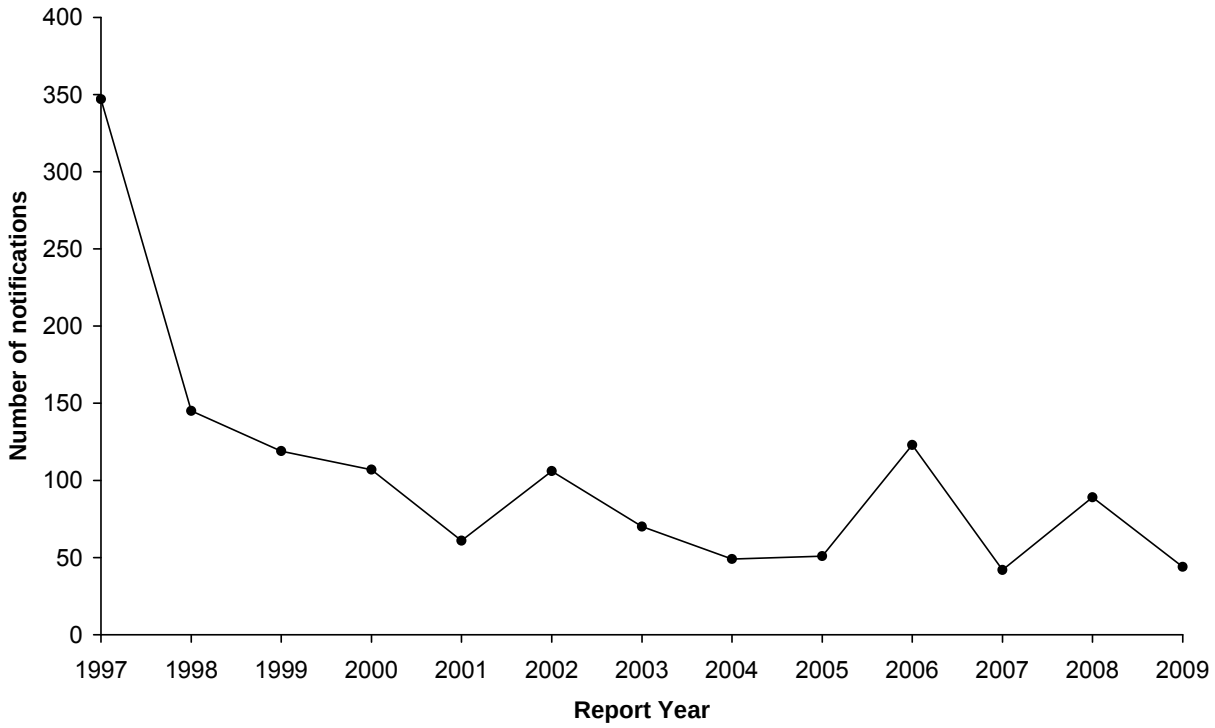
The ICD-10 code B15 was used to extract hepatitis A hospitalisation data from the MoH NMDS database. Of the 24 hospital admissions (0.6 admissions per 100 000 population) recorded in 2009, 17 were reported with hepatitis A as the primary diagnosis and seven with hepatitis A as another relevant diagnosis.

4.9.3 Notifiable disease data

4.9.3.1 Annual notification trend

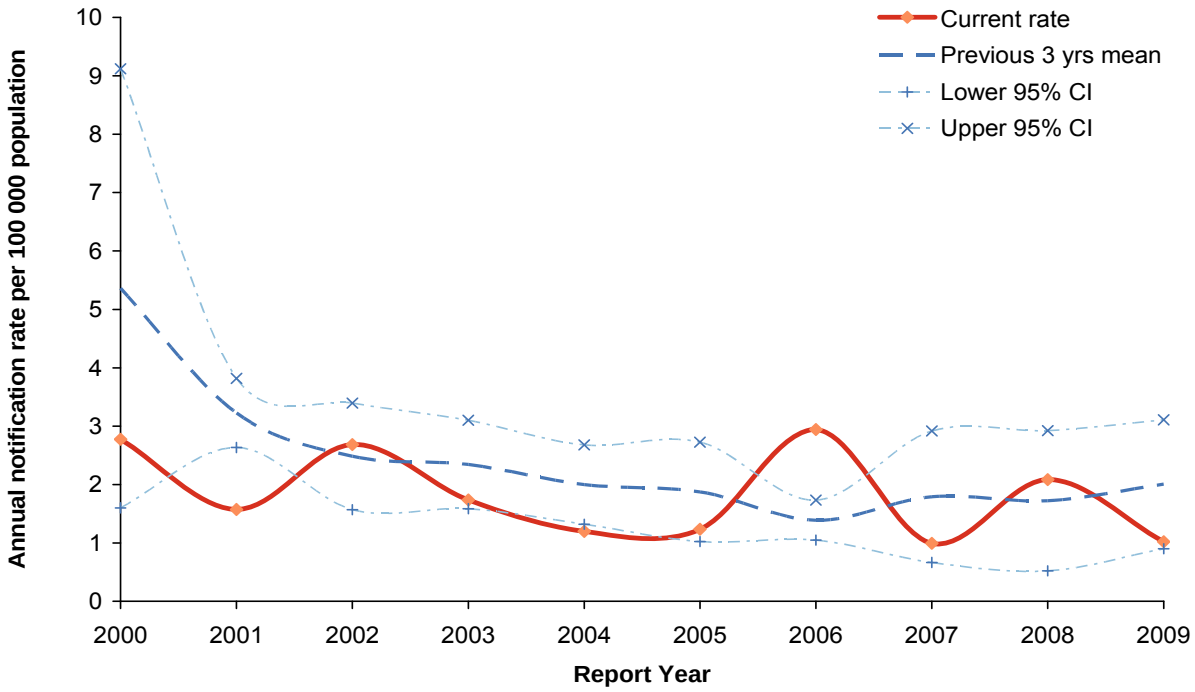
Over the last thirteen years, there has been an overall downward trend in the number of notifications of hepatitis A, although a local increase in notifications was observed in 2002, 2006 and again in 2008 (Figure 27).

Figure 27: Hepatitis A notifications by year, 1997-2009



Hepatitis A notification rates varied throughout the ten-year period, 2000-2009 (Figure 28). The notification rate trend is very similar to the corresponding annual notification trend, showing peaks in 2002, 2006 and 2008. The highest hepatitis A notification rate was recorded in 2006 (2.9 per 100 000 population).

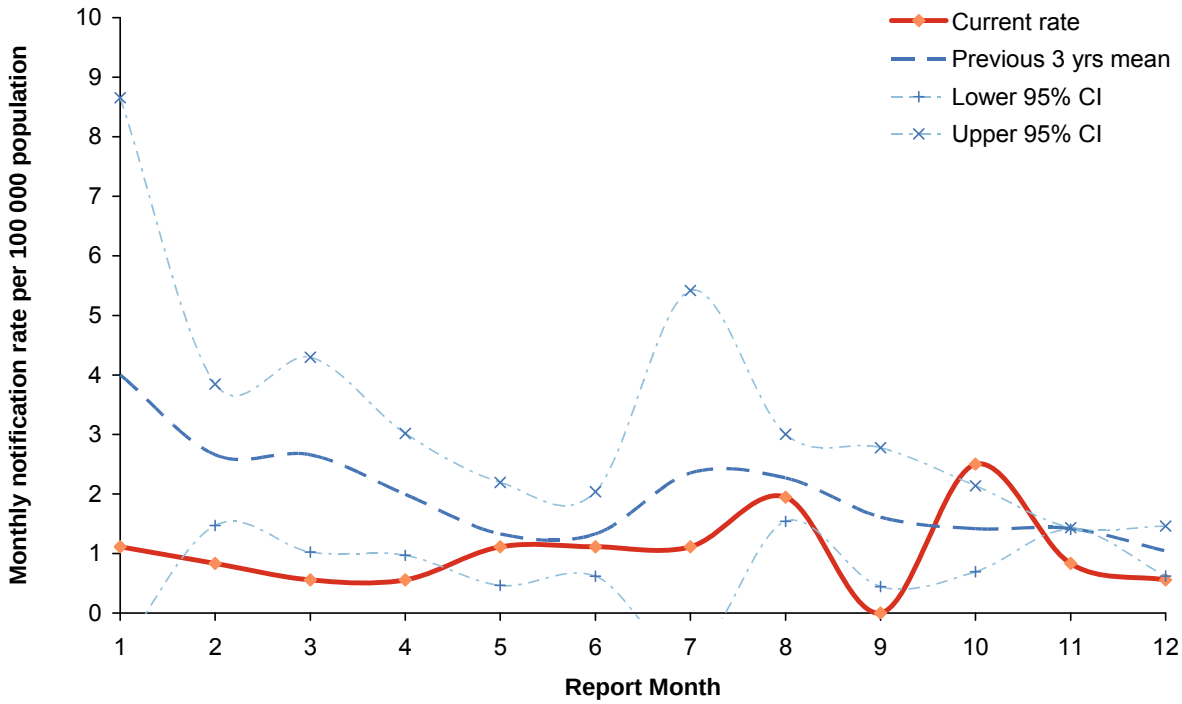
Figure 28: Hepatitis A notification rate by year, 2000-2009



4.9.3.2 Seasonality

There was no strong seasonal pattern in the population rate of hepatitis A notifications reported by month either historically or in 2009.

Figure 29: Hepatitis A monthly rate (annualised) for 2009



4.9.3.3 Age and sex distribution of hepatitis A cases

In 2009, the hepatitis A notification rate was higher for males than females, whereas the hospitalisation rate was similar for both genders (Table 30).

Table 30: Hepatitis A cases by sex, 2009

Sex	EpiSurv notifications		Morbidity[a]		Deaths recorded in EpiSurv
	No.	Rate[b]	No.	Rate[b]	No.
Male	26	1.2	13	0.6	
Female	18	0.8	11	0.5	
Unknown	0				
Total	44	1.0	24	0.6	

[a] MoH morbidity data for hospital admissions
[b] per 100 000 of population

The age-specific hepatitis A notification rate in 2009 was highest for those aged 10 to 14 years (2.0 per 100 000 population, 6 cases). The notification and hospitalisation rates were not defined for most age groups due to the small number of cases.

Table 31: Hepatitis A cases by age group, 2009

Age group	EpiSurv notifications		Morbidity[a]		Deaths recorded in EpiSurv
	No.	Rate[b]	No.	Rate[b]	No.
<1	0	-	0	-	
1 to 4	2	-	0	-	
5 to 9	4	-	1	-	
10 to 14	6	2.0	1	-	
15 to 19	3	-	1	-	
20 to 29	6	1.0	4	-	
30 to 39	4	-	4	-	
40 to 49	9	1.4	4	-	
50 to 59	6	1.1	5	0.9	
60 to 69	4	-	2	-	
70+	0	-	2	-	
Unknown	0				
Total	44	1.0	24	0.6	

[a] MoH morbidity data for hospital admissions
[b] per 100 000 of population. Where fewer than five cases have been reported a rate has not been calculated.

4.9.3.4 Risk Factors Reported

The most commonly reported risk factor for hepatitis A in 2009 was overseas travel during the incubation period (75.0%) (Table 32).

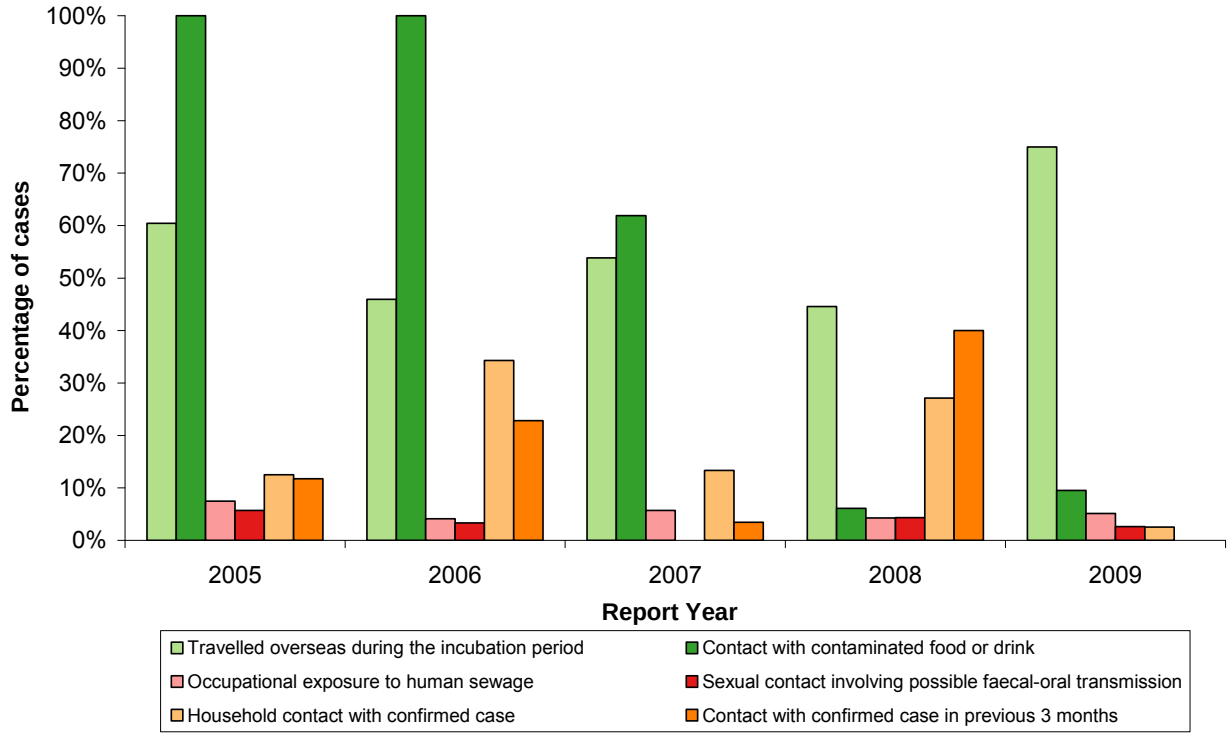
Table 32: Exposure to risk factors associated with hepatitis A, 2009

	Notifications			
Risk Factor	**Yes**	**No**	**Unknown**	**%[a]**
Travelled overseas during the incubation period	33	11	0	75.0
Contact with contaminated food or drink	2	19	23	9.5
Occupational exposure to human sewage	2	37	5	5.1
Sexual contact involving possible faecal-oral transmission	1	37	6	2.6
Household contact with confirmed case	1	38	5	2.6
Contact with confirmed case in previous 3 months	0	34	10	0.0

[a]Percentage refers to the cases that answered "yes" out of the total number of cases for which this information was supplied. Cases may have more than one risk factor recorded.

Between 2005 and 2007 the risk factors associated with hepatitis A cases generally occurred in the same order of importance with a high proportion of cases reporting contact with contaminated food or drink (Figure 30). During 2008 and 2009, contact with contaminated food or drink was identified as a risk factor by only a small proportion of cases, instead overseas travel during the incubation period was the most frequently identified risk factor. Since 2005, 44.6% to 75.0% of cases each year have reported overseas travel during the incubation period of the disease.

Figure 30: Hepatitis A risk factors by percentage of cases and year, 2005-2009



4.9.3.5 Estimate of travel-related cases

For cases where information on travel was provided, 75.0% (95%CI 59.7-86.8%) had travelled overseas during the incubation period. Assuming that the cases for which travel information was provided were representative of all hepatitis A cases, a Poisson distribution can be used to estimate the total number of potentially travel related cases of hepatitis A in 2009. The resultant distribution has a mean of 33 cases (95% CI 21-44).

If data from the last four years are considered the estimated proportion of cases travelling overseas within the incubation period of the organism is 52.0% (95% CI 45.9-58.0%).

4.9.4 Outbreaks reported as caused by hepatitis A virus

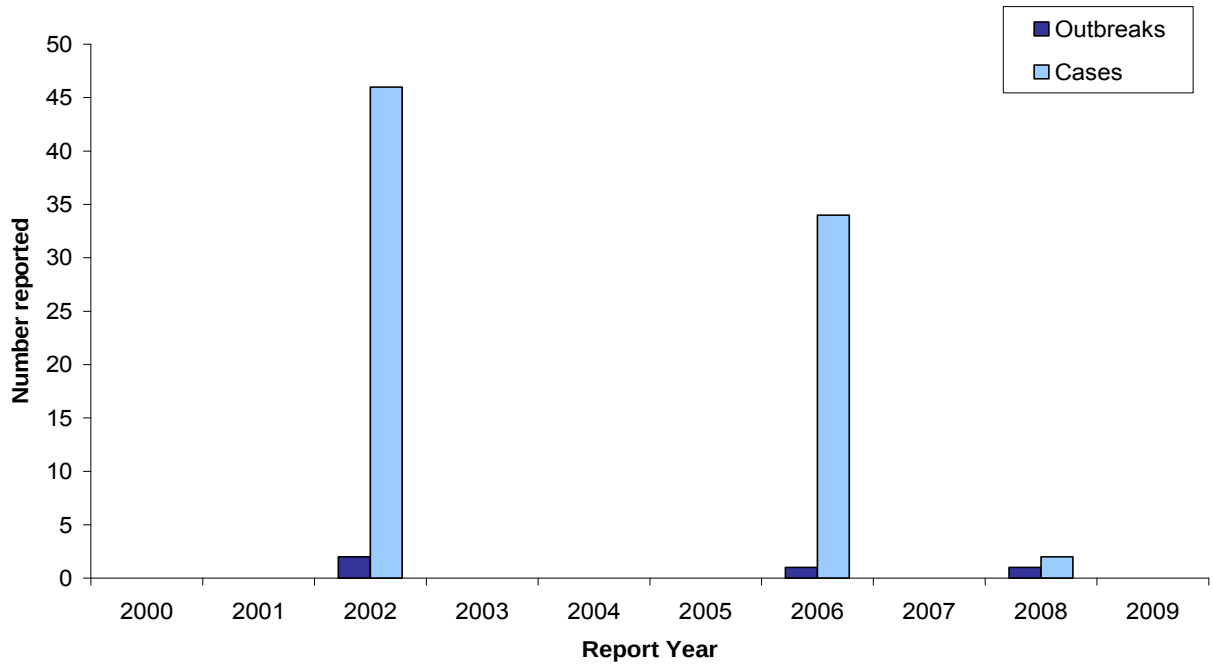
No foodborne hepatitis A virus outbreaks were reported in 2009 (Table 33).

Table 33: Hepatitis A virus outbreaks reported, 2009

Measure (No.)	Foodborne hepatitis A virus outbreaks	All hepatitis A virus outbreaks
Outbreaks	0	1
Cases	0	2
Hospitalised cases	0	0

Foodborne hepatitis A virus outbreaks are rare with only four reported in the period 2000 to 2009 (in 2002, 2006 and 2008) (Figure 31). Although occurring infrequently, foodborne outbreaks of hepatitis A virus can be associated with many cases, although this was not so for the food-associated outbreak in 2008.

Figure 31: Foodborne hepatitis A virus outbreaks and associated cases reported by year, 2000–2009



4.9.4.1 Details of food-associated outbreaks

No foodborne hepatitis A outbreaks were reported in 2009

4.9.5 Laboratory investigation of samples from suspected foodborne outbreaks

During investigations of suspected foodborne illness outbreaks by ESR's Public Health Laboratory, no samples were found to contain hepatitis A virus.

4.9.6 Relevant New Zealand studies and publications

Nil.

4.9.7 Relevant regulatory developments

Nil.

4.10 Histamine (Scombroid) Fish Poisoning

4.10.1 Case definition

Clinical description:	Tingling and burning sensation around mouth, facial flushing, sweating, nausea and vomiting, headache, palpitations, dizziness and rash
Laboratory test for diagnosis:	Detection of histamine levels ≥ 50mg/100 g fish muscle
Case classification:	Not applicable

4.10.2 Histamine (scombroid) fish poisoning cases reported in 2009 by data source

Four cases of histamine (scombroid) fish poisoning and no resulting deaths were reported in EpiSurv during 2009.

The ICD-10 code T61.1 was used to extract scombroid fish poisoning hospitalisation data from the MoH NMDS database. Of the 3 hospital admissions recorded in 2009, all were reported with scombroid fish poisoning as the primary diagnosis.

4.10.3 Outbreaks reported as caused by histamine (scombroid) fish poisoning

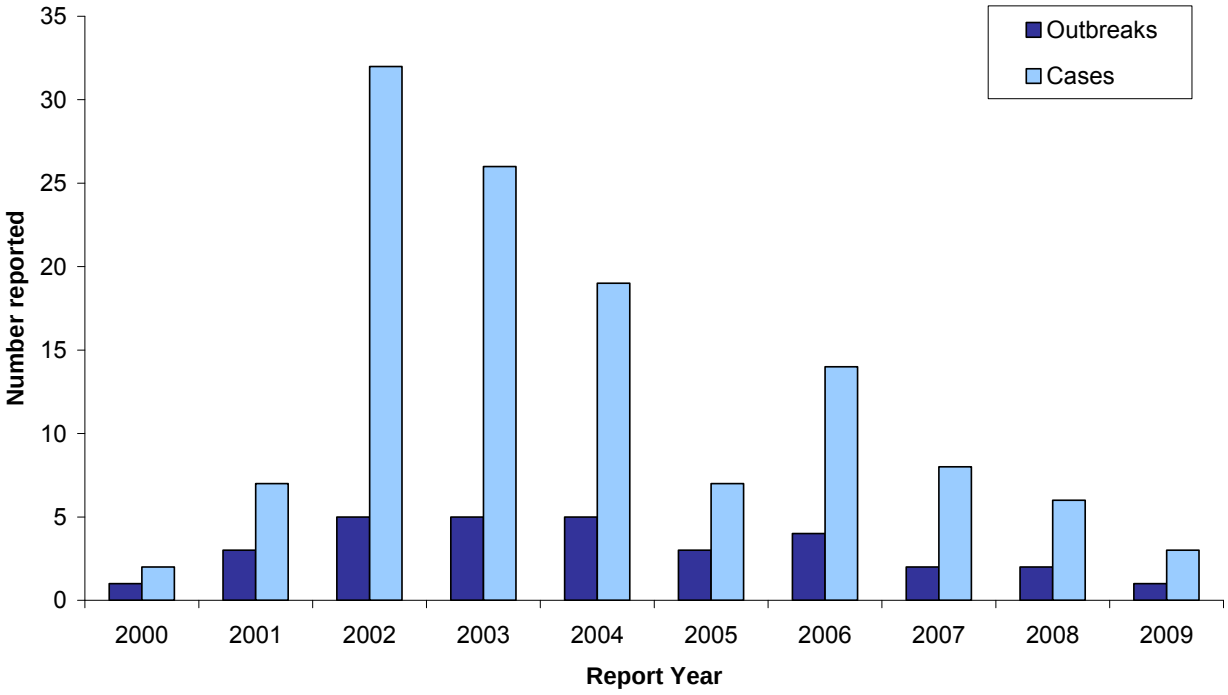
One histamine (scombroid) fish poisoning outbreak was reported in 2009 involving three associated cases, with no cases hospitalised (Table 34). The outbreak reported foodborne transmission.

Table 34: Histamine (scombroid) fish poisoning outbreaks reported, 2009

Measure (No.)	Foodborne histamine fish poisoning outbreaks	All histamine fish poisoning outbreaks
Outbreaks	1	1
Cases	3	3
Hospitalised cases	0	0

Between 2000 and 2009 the number of foodborne histamine (scombroid) fish poisoning outbreaks reported each year has ranged from one to five (Figure 32). The highest number of outbreaks was reported between 2002 and 2004 (5 outbreaks reported each year) and the highest total number of associated cases was reported in 2002 (32 cases). Since 2002, the total number of cases associated with the outbreaks has generally decreased.

Figure 32: Histamine (scombroid) fish poisoning outbreaks and associated cases reported by year, 2000 – 2009



4.10.3.1 Details of food-associated outbreaks

Table 35 contains details of the histamine poisoning outbreak reported in 2009.

Table 35: Details of food-associated histamine poisoning outbreaks, 2009

Public Health Unit (Month)	Suspected vehicle	Setting	Number ill	Confirmation
Auckland (February)	King fish	Restaurant/Café	3P	6

C = confirmed, P = probable
Confirmation:
1 = Environmental investigation – identified critical control point failures linked to implicated source
2 = Epidemiological – case had history of exposure to implicated source
3 = Epidemiological – case control or cohort study showed elevated risk for cases to implicated source
4 = Laboratory – pathogen suspected to have caused illness identified in food handler
5 = Laboratory – pathogen suspected to have caused illness identified in implicated source (food)
6 = No evidence
7 = Other evidence

Histamine poisoning is virtually always associated with consumption of scombroid fish species. This significantly assists identification of causal foods and evidence linking outbreaks to foods is consequently strong in most outbreaks.

4.10.3.2 Laboratory investigation of samples from suspected foodborne outbreaks

During investigations of suspected foodborne illness outbreaks by ESR's Food Chemistry Laboratory, analyses were carried out on a fish sample from one investigation. The histamine concentration in the fish sample analysed was 970 mg/kg (97 mg/100 g). This is sufficiently high to cause histamine poisoning.

4.10.4 Relevant New Zealand studies and publications

Nil.

4.10.5 Relevant regulatory developments

In August 2009, changes were made to the Imported Food Requirements for Prescribed Foods[3]. For fish species susceptible to production of histamine changes included:

- Products imported into New Zealand from Australia are not subject to NZFSA import clearance requirements. Importers do not need to apply for a Single Use Permit for clearance. This applies to food produced in Australia and to food imported into Australia.

- Importers still have a responsibility under the Food (Importer General Requirements) Standard 2008 to ensure that imports of histamine susceptible fish do not have excessive histamine levels. Selected species of fish (e.g. tuna, mackerel, amberjack (yellowtail kingfish), mahi mahi, bluefish, sardine including pilchard and herring) are more susceptible to microbiological spoilage and the production of histamine. Amines, including histamines, are only produced during temperature abuse or spoilage. Histamines are heat stable and are not destroyed during cooking or canning processes. Good manufacturing practices,

[3] http://www.nzfsa.govt.nz/importing/documents/changes-to-importing-system/summary-of-changes-for-ifrs/

particularly maintaining products at chilled temperatures, reduce the likelihood of histamine production.

- Importers should confirm appropriate handling of the product and additional assurances to satisfy themselves that the supplier is managing the risks. For example: supplier assurance programme, a written assurance for each consignment from their supplier that the product does not have excess levels of histamine (less than 200 mg/kg), etc.
- Products and tariff codes have been reviewed, revised and clarified to ensure that high risk products are appropriately targeted.

4.11 Listeriosis

Summary data for listeriosis in 2009 are given in Table 36.

Table 36: Summary surveillance data for listeriosis, 2009

Parameter	Value in 2009	Section reference
Number of cases	28	4.11.2
Rate (per 100,000)	0.6	4.11.2
Hospitalisations (%)	28 (100.0%)	4.11.2
Deaths (%)	4 (14.3%)	4.11.2
Estimated travel-related cases (%)	3 (9.1%)	4.11.3.4
Estimated food-related cases (%)*	21 (84.9%)	4.11.2

* For estimation of food-related cases it was assumed that the proportions derived from expert consultation would exclude travel-related cases

4.11.1 Case definition

Clinical description: An infection which produces several clinical syndromes including stillbirths, listeriosis of the newborn, meningitis, bacteraemia, or localised infections. Pregnant women, the immunosuppressed and the frail elderly are at greatest risk

Laboratory test for diagnosis: Isolation of *Listeria monocytogenes* from a site that is normally sterile, including the foetal gastrointestinal tract

Case classification:
Probable Not applicable
Confirmed A clinically compatible illness that is laboratory confirmed

4.11.2 Listeriosis cases reported in 2009 by data source

During 2009, 28 notifications (0.6 cases per 100 000 population) of listeriosis were reported in EpiSurv, of which 10 were perinatal. Twenty-nine cultures were received by the ESR Special Bacteriology Laboratory.

The ICD-10 code A32 was used to extract listeriosis hospitalisation data from the MoH NMDS database. Of the 28 hospital admissions (0.6 admissions per 100 000 population) recorded in 2009, 11 were reported with listeriosis as the primary diagnosis and 17 with listeriosis as another relevant diagnosis.

Two deaths resulting from non-perinatal listeriosis and two from perinatal listeriosis were recorded in EpiSurv in 2009.

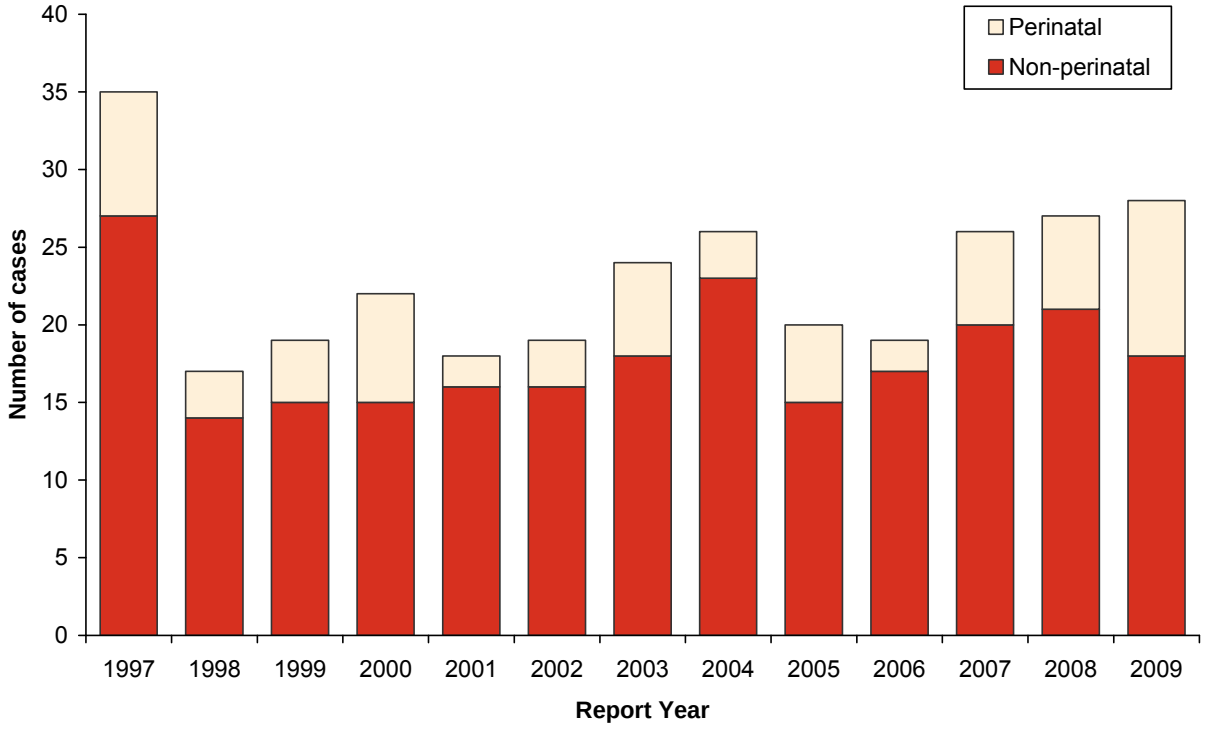
It has been estimated by expert consultation that 85% (minimum = 78%, maximum = 92%) of listeriosis incidence is due to foodborne transmission. It was further estimated that approximately 50% of foodborne transmission was due to consumption of ready-to-eat meats, while approximately 7% was due to ice cream consumption.

4.11.3 Notifiable disease data

4.11.3.1 Annual notification trend

The number of listeriosis notifications has generally increased since 2006 (Figure 33). The highest number of notifications was reported in 1997 (35 cases). The highest number of perinatal cases was reported in 2009 (10 cases).

Figure 33: Listeriosis non-perinatal and perinatal notifications by year, 1997-2009



4.11.3.2 Age and sex distribution of listeriosis cases

In 2009, the number and rate of notifications for listeriosis were higher for females compared to males. The number of hospitalisations for females was also higher than for males (Table 37).

Table 37: Listeriosis cases by sex, 2009

Sex	EpiSurv notifications		Morbidity[a]		Deaths recorded in EpiSurv[b]
	No.	Rate[c]	No.	Rate[c]	No.
Male	6	0.3	9	0.4	1
Female	22	1.0	19	0.9	1
Total	28	0.6	28	0.6	2

[a] MoH morbidity data for hospital admissions
[b] Perinatal cases are recorded in terms of the mother's demography and perinatal deaths are not recorded in this table
[c] per 100 000 of population

In 2009, the age specific listeriosis notification rates were highest in the 70+ years age group (2.6 per 100 000 population, 10 cases) and the 20 to 29 years age group (0.9 per 100 000, 5 cases) (Table 38). The highest hospitalisation rates were in the 70+ years age group. The notification and hospitalisation rates were not defined for most age groups due to the small number of cases.

Table 38: Listeriosis cases by age group, 2009

Age group	EpiSurv notifications		Morbidity[a]		Deaths recorded in EpiSurv[b]
	No.	Rate[c]	No.	Rate[c]	No.
<1	0	-	2	-	
1 to 4	0	-	0	-	
5 to 9	0	-	0	-	
10 to 14	1	-	2	-	
15 to 19	2	-	1	-	
20 to 29	5	0.9	7	1.2	
30 to 39	4	-	2	-	
40 to 49	1	-	0	-	
50 to 59	2	-	2	-	
60 to 69	3	-	3	-	
70+	10	2.6	9	2.4	2
Total	28	0.6	28	0.6	2

[a] MoH morbidity data for hospital admissions
[b] Perinatal cases are recorded in terms of the mother's demography and perinatal deaths are not recorded in this table
[c] per 100 000 of population. Where fewer than five cases have been reported a rate has not been calculated.

4.11.3.3 Risk Factors Reported

During 2009, the most common risk factors reported for listeriosis were an underlying illness (76.5%) and hospital admission for another illness (46.2%) (Table 39).

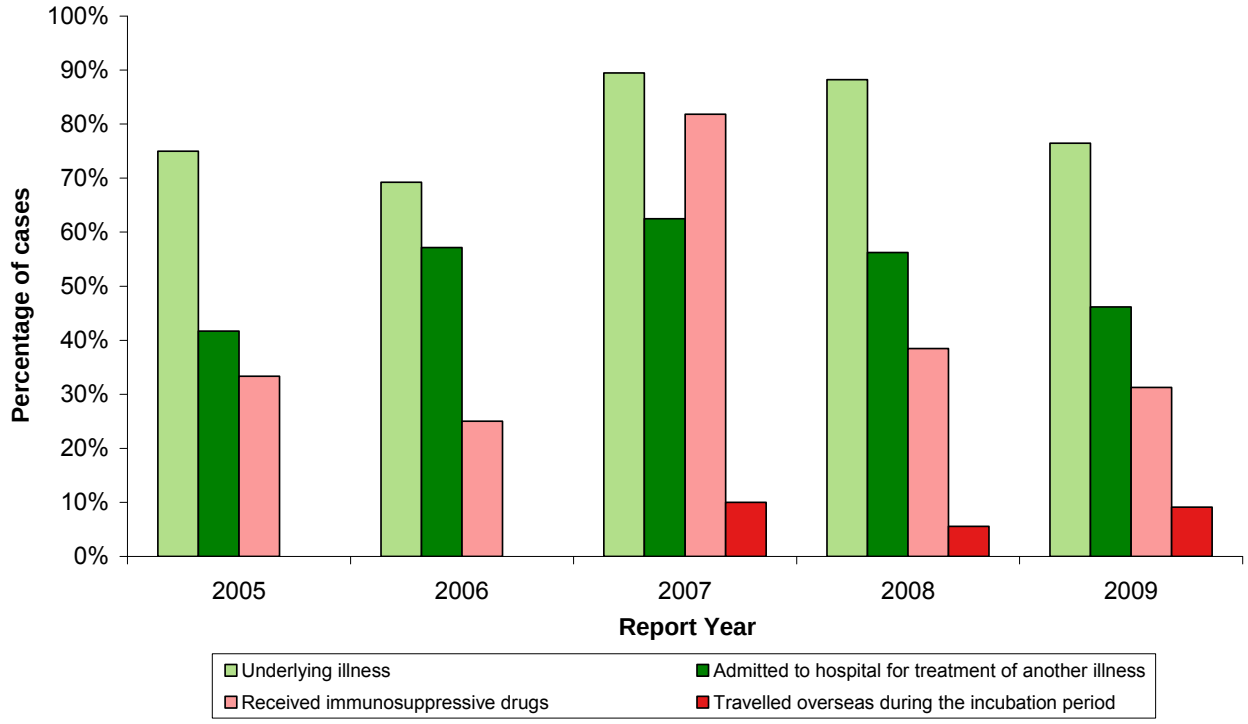
Table 39: Exposure to risk factors associated with listeriosis, 2009

	Notifications			
Risk Factor	**Yes**	**No**	**Unknown**	**%[a]**
Underlying illness	13	4	1	76.5
Admitted to hospital for treatment of another illness	6	7	5	46.2
Received immunosuppressive drugs	5	11	2	31.3
Travelled overseas during the incubation period	1	10	7	9.1

[a]Percentage refers to the cases that answered "yes" out of the total number of cases for which this information was supplied. Cases may have more than one risk factor recorded. Perinatal cases are excluded from this analysis.

Between 2005 and 2009 the risk factors associated with listeriosis cases have generally occurred in a similar order of importance each year (Figure 34). Every year an underlying illness was the risk factor most commonly reported for listeriosis.

Figure 34: Listeriosis risk factors by percentage of cases and year, 2005-2009



4.11.3.4 Estimate of travel-related cases

For cases where information on travel was provided, 9.1% (95%CI 0.2-41.3%) had travelled overseas during the incubation period. Assuming that the cases for which travel information was provided were representative of all listeriosis cases, a Poisson distribution can be used to estimate

the total number of potentially travel related cases of listeriosis in 2009. The resultant distribution has a mean of 3 cases (95% CI 0-9).

4.11.4 Outbreaks reported as caused by *Listeria* spp.

One *Listeria monocytogenes* outbreak was reported in 2009 involving two associated cases, with no cases hospitalised (Table 40). The outbreak reported foodborne transmission.

Table 40: *Listeria* outbreaks reported, 2009

Measure (No.)	Foodborne *Listeria* outbreaks	All *Listeria* outbreaks
Outbreaks	1	1
Cases	2	2
Hospitalised cases	0	0

The outbreak reported in 2009 is the only *Listeria* outbreak to be reported for the period 2000 to 2009.

4.11.4.1 Details of food-associated outbreaks

Table 41 contains details of the one food-associated *Listeria* outbreak reported in 2009.

Table 41: Details of food-associated *Listeria* outbreaks, 2009

Public Health Unit (Month)	Suspected vehicle	Setting	Number ill	Confirmation
Auckland (July)	Unknown	Takeaway, Supermarket/ Delicatessen, Other food outlet	2C	6

C = confirmed, P = probable
Confirmation:
1 = Environmental investigation – identified critical control point failures linked to implicated source
2 = Epidemiological – case had history of exposure to implicated source
3 = Epidemiological – case control or cohort study showed elevated risk for cases to implicated source
4 = Laboratory – pathogen suspected to have caused illness identified in food handler
5 = Laboratory – pathogen suspected to have caused illness identified in implicated source (food)
6 = No evidence
7 = Other evidence

While this outbreak was reported as food-associated in EpiSurv, the suspected vehicle is unknown and no evidence was available linking the outbreak to an implicated source.

4.11.4.2 Laboratory investigation of samples from suspected foodborne outbreaks

During investigations of suspected foodborne illness outbreaks by ESR's Public Health Laboratory, *Listeria monocytogenes* was not isolated from any samples. *Listeria innocua* was isolated from a surface swab from a refrigerator in the home of a notified listeriosis cases.

4.11.5 Recent Surveys

Nil.

4.11.6 Relevant New Zealand studies and publications

Nil.

4.11.7 Relevant regulatory developments

In March 2009 NZFSA released their *Listeria monocytogenes* Risk Management Strategy 2008-2013:
http://www.nzfsa.govt.nz/foodborne-illness/listeria/strategy.htm

This document states that the strategy will:

- Ensure that risk management options for the control of *L. monocytogenes* are effective and applied consistently across all food businesses;
- Take account of international developments in *L. monocytogenes* risk management through involvement in international fora and collaborations;
- Provide enhanced and effective information to all stakeholders for reducing the potential for *L. monocytogenes* contamination of food and exposure of consumers to potentially contaminated food;
- Document a process that will monitor and review progress of the strategy to meet the SOI (Statement of Intent) performance target; and
- Identify and prioritise research needed to inform and support *L. monocytogenes* risk management options applied and proposed.

The SOI performance target is "no increase in reported incidence of foodborne listeriosis after five years".

The objectives of the strategy are:

- To achieve no increase in human foodborne listeriosis cases;
- To engage with industry, other stakeholders and consumers in order to ensure that any outcomes developed are practical, feasible and cost effective;
- To effectively communicate the strategy and outcomes to all stakeholders (including consumers);
- To make well informed risk management decisions on appropriate control measures and their implementation; and
- To design and implement an ongoing monitoring and review programme to assess the effectiveness of risk management decisions.

NZFSA has developed a Code of Practice for production of processed meats, which was the subject of a consultation process during 2009 (http://www.nzfsa.govt.nz/consultation/processed-meat-cop-part1-4/index.htm). It is envisaged that this will be used by processors operating a Food Safety Plan under the Food Act 1981, those operating a Risk Management Plan under the Animal Products Act 1999, and those operating under the Food Hygiene Regulation 1974.

The draft Code includes provision for an environmental monitoring programme for *Listeria*. It also states that "Cooked cured/salted meat products must meet the microbiological limits given in the Food Standards Code, Standard 1.6.1." (see below) and "When cooking is used to control pathogens in ready-to-eat (RTE) products, the cooking process must achieve a 6 decimal reduction of *Listeria monocytogenes* (a 6D process).". For such cooking, times and temperatures are recommended, although alternative approaches may be used provided they are validated by the

processor and approved by the NZFSA. The HACCP plans included with the consultation documents specifically address the potential for *Listeria* contamination during processing.

4.12 Norovirus Infection

4.12.1 Case definition

Clinical description:	Gastroenteritis usually lasting 12-60 hours
Laboratory test for diagnosis:	Detection of norovirus in faecal or vomit specimen or leftover food
Case classification:	
Probable	A clinically compatible illness that is either a contact of a confirmed case of the same disease, or has had contact with the same common source i.e., is part of an identified common source outbreak
Confirmed	A clinically compatible illness that is laboratory confirmed

4.12.2 Norovirus infection cases reported in 2009 by data source

During 2009, 250 notifications (5.8 cases per 100 000 population) of norovirus and no resulting deaths were reported in EpiSurv.

The ICD-10 code A08.1 was used to extract norovirus infection hospitalisation data from the MoH NMDS database. Of the 319 hospital admissions (7.4 admissions per 100 000 population) recorded in 2009, 64 were reported with norovirus infection as the primary diagnosis and 255 with norovirus infection as another relevant diagnosis.

An expert consultation estimated that 40% of norovirus infections were due to foodborne transmission and of these 40% were due to consumption of molluscan shellfish.

4.12.3 Outbreaks reported as caused by norovirus

During 2009, there were 270 norovirus outbreaks reported in EpiSurv and of these 29 were associated with a suspected or known foodborne source (Table 42). In total, 349 cases were associated with these foodborne outbreaks.

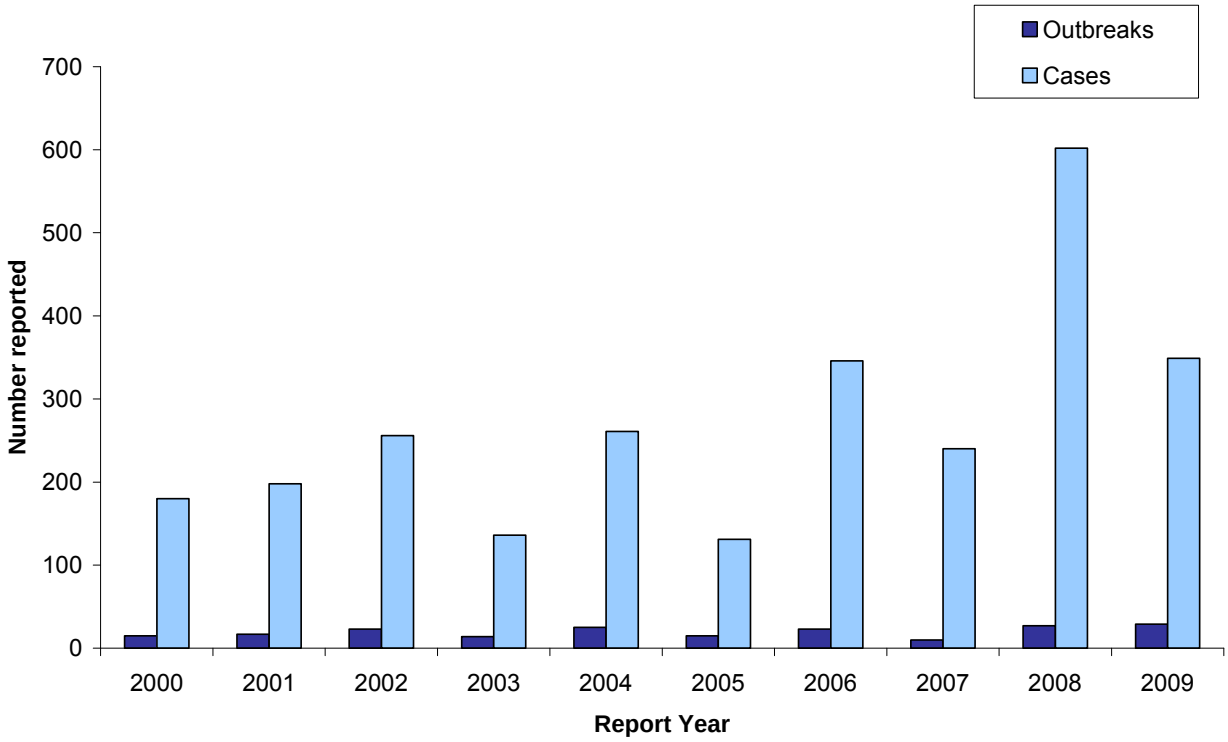
Table 42: Norovirus outbreaks reported, 2009

Measure (No.)	Foodborne norovirus outbreaks	All norovirus outbreaks
Outbreaks	29	270
Cases	349	7 116
Hospitalised cases	5	243

The number of foodborne outbreaks in 2009 was greater than in any of the prior nine years and the number of associated cases was the second highest reported (Figure 35). From 2000 to 2009 the number of foodborne norovirus outbreaks reported each year ranged from 10 (in 2007) to 29 (in

2009). The total number of cases associated with these outbreaks had ranged from 131 (in 2005) to 602 (in 2008).

Figure 35: Foodborne norovirus outbreaks and associated cases reported by year, 2000–2009



4.12.3.1 Details of food-associated outbreaks

Table 43 contains details of the 29 food–associated norovirus outbreaks reported in 2009.

Table 43: Details of food-associated norovirus outbreaks, 2009

Public Health Unit (Month)	Suspected vehicle	Setting	Number ill	Confirmation
Auckland (July)	Oysters	Workplace	5C, 12P	5
Auckland (July)	Oysters	Restaurant/Café	1C, 1P	6
Auckland (August)	Chicken, potato and gravy	Restaurant/Café	2C, 2P	2
Auckland (August)	Sushi, sauces, sausage rolls, salmon sandwiches	Caterers, Home, Supermarket/ Delicatessen	2C, 15P	2
Auckland (August)	Fish and chips, sausages, spring rolls	Takeaway	5C, 6P	7
Auckland (September)	Ice cream	Takeaway	1C, 1P	Unknown
Auckland (September)	Unknown	Home, Takeaway	2C	2
Auckland (September)	Unknown	Rest home	1C, 28P	6
Auckland (October)	Salmon meal, potato bake	Restaurant/Café	2C	Unknown
Auckland (October)	Chicken, potato and gravy	Restaurant/Café	1C, 1P	6

Public Health Unit (Month)	Suspected vehicle	Setting	Number ill	Confirmation
Auckland (November)	Oysters	Restaurant/Café	1C, 1P	6
Auckland (November)	Salmon meal	Restaurant/Café	1C, 1P	2
Auckland (November)	Oysters	Restaurant/Café, Home	1C, 1P	6
Auckland (November)	Oysters	Restaurant/Café	3C	2
Auckland (December)	Chicken roll	Home, Restaurant/Café	3C, 2P	6
Auckland (December)	Pizza, stir fry noodles, hot chips, mashed potatoes, dessert	Home, Restaurant/Café	2P	6
Auckland (December)	Infected food handler	Rehab clinic	5C, 15P	Unknown
Canterbury (December)	Unknown	Restaurant/Café	1C, 3P	2
Nelson (September)	Infected food handler	Golf Club	6C, 20P	2, 4
Otago (March)	Unknown	Childcare centre	2C, 20P	2
Otago (April)	Assorted sandwiches	Restaurant/Café	5C, 22P	2, 3
Tauranga (December)	Unknown	Restaurant/Café, Workplace	4C	6
Waikato (July)	Unknown	Rest home	62C	3
Waikato (August)	Oysters	Restaurant/Café	3C	2, 3, 5
Waikato (December)	Unknown	Home	1C, 6P	6
Wellington (September)	Oysters	Hotel/Motel	6C, 6P	3, 7
West Coast (September)	Unknown	Rest home	1C, 19P	3
West Coast (September)	Unknown	Hospital (continuing care)	1C, 21P	1, 3
West Coast (October)	Unknown	Hospital (continuing care)	1C, 15P	2

C = confirmed, P = probable
Confirmation:
1 = Environmental investigation – identified critical control point failures linked to implicated source
2 = Epidemiological – case had history of exposure to implicated source
3 = Epidemiological – case control or cohort study showed elevated risk for cases to implicated source
4 = Laboratory – pathogen suspected to have caused illness identified in food handler
5 = Laboratory – pathogen suspected to have caused illness identified in implicated source (food)
6 = No evidence
7 = Other evidence

Oysters were implicated in approximately one-quarter of norovirus-associated outbreaks (7/29; 24%), linked to three different oyster suppliers. There was occasionally stronger evidence implicating oysters (e.g. organism detected in suspect food) than for other food vehicles, due to the availability of methods to detect norovirus in oysters. Such methods are not generally available for other foods.

4.12.3.2 Laboratory investigation of samples from suspected foodborne outbreaks

During investigations of illness outbreaks caused by potentially foodborne organisms by ESR's Public Health Laboratory, norovirus was detected in faecal samples from 78 investigations. Norovirus was detected in two oyster samples and one mussel sample from three investigations. However, the type detected in the mussel sample (NVG I) differed from the type detected in the associated faecal sample (NVG II). Norovirus was also detected in two water samples associated with outbreaks.

A diverse range of foods were implicated in these investigations, although in many investigations no food was implicated and some outbreaks, including several large outbreaks in institutional settings (rest homes, childcare centres), are likely to have been due to person-to-person transmission.

4.12.4 Relevant New Zealand studies and publications

4.12.4.1 Reports

A one-year study of the microbiological and virological quality of shellfish in Tauranga harbour was carried out (Greening, 2009). Results showed that viruses were present in shellfish on many occasions during the year. Overall noroviruses were detected in 23/72 (32%; 95th percentile confidence interval 21-44%) monthly surveillance samples. Norovirus GI strains were less frequently detected than norovirus GII strains. Adenoviruses were only detected on 7/72 (9.7%; 95th percentile confidence interval 4-19%) occasions during the surveillance study and on 9 occasions following pollution events.

Noroviruses were detected in 19/25 (76%; 95th percentile confidence interval 55-91%) shellfish samples and one water sample following a point source sewage contamination event, but in only 13/49 (27%; 95th percentile confidence interval 15-41%) of shellfish samples following rainfall events. Human-associated F-RNA bacteriophage genogroups II and III were commonly identified in samples following both the point source event (15/25, 60%; 95th percentile confidence interval 39-79%) and the rainfall event (32/49, 65%; 95th percentile confidence interval 50-78%) whereas animal-associated F-RNA bacteriophage genogroup I was more commonly detected in shellfish following the rainfall event (22/49, 45%; 95th percentile confidence interval 31-60%) than the point source event (4/25, 16%; 95th percentile confidence interval 5-36%). No genogroup IV F-RNA bacteriophage were detected during the study.

4.12.5 Relevant regulatory developments

Nil.

4.13 Salmonellosis

Summary data for salmonellosis in 2009 are given in Table 44.

Table 44: Summary surveillance data for salmonellosis, 2009

Parameter	Value in 2009	Section reference
Number of cases	1 129	4.13.2
Rate (per 100,000)	26.2	4.13.2
Hospitalisations (%)	158	4.13.2
Deaths (%)	1 (0.09%)	4.13.2
Estimated travel-related cases (%)	186 (16.4%)	4.13.3.6
Estimated food-related cases (%)*	572 (60.7%)	4.13.2

* For estimation of food-related cases it was assumed that the proportions derived from expert consultation would exclude travel-related cases

4.13.1 Case definition

Clinical description:	Salmonellosis presents as gastroenteritis. Asymptomatic infections may occur
Laboratory test for diagnosis:	Isolation of *Salmonella* species (excluding *S.* Typhi) from any clinical specimen
Case classification:	
Probable	A clinically compatible illness that is either a contact of a confirmed case of the same disease, or has had contact with the same common source i.e., is part of an identified common source outbreak
Confirmed	A clinically compatible illness that is laboratory confirmed

4.13.2 Salmonellosis cases reported in 2009 by data source

The salmonellosis cases presented here exclude disease caused by *S.* Paratyphi and *S.* Typhi.

During 2009, 1 129 notifications (26.2 cases per 100 000 population) of salmonellosis were reported in EpiSurv. The Enteric Reference Laboratory at ESR confirmed and reported 1 122 *Salmonella* isolates (26.0 cases per 100 000).

The ICD-10 code A02.0 was used to extract salmonellosis hospitalisation data from the MoH NMDS database. Of the 158 hospital admissions (3.7 admissions per 100 000 population) recorded in 2009, 130 were reported with salmonellosis as the primary diagnosis and 28 with salmonellosis as another relevant diagnosis.

One death resulting from salmonellosis was recorded in EpiSurv in 2009.

It has been estimated by expert consultation that 61% (minimum = 45%, maximum = 69%) of salmonellosis incidence is due to foodborne transmission. It was further estimated that 36% of foodborne transmission was due to transmission via poultry.

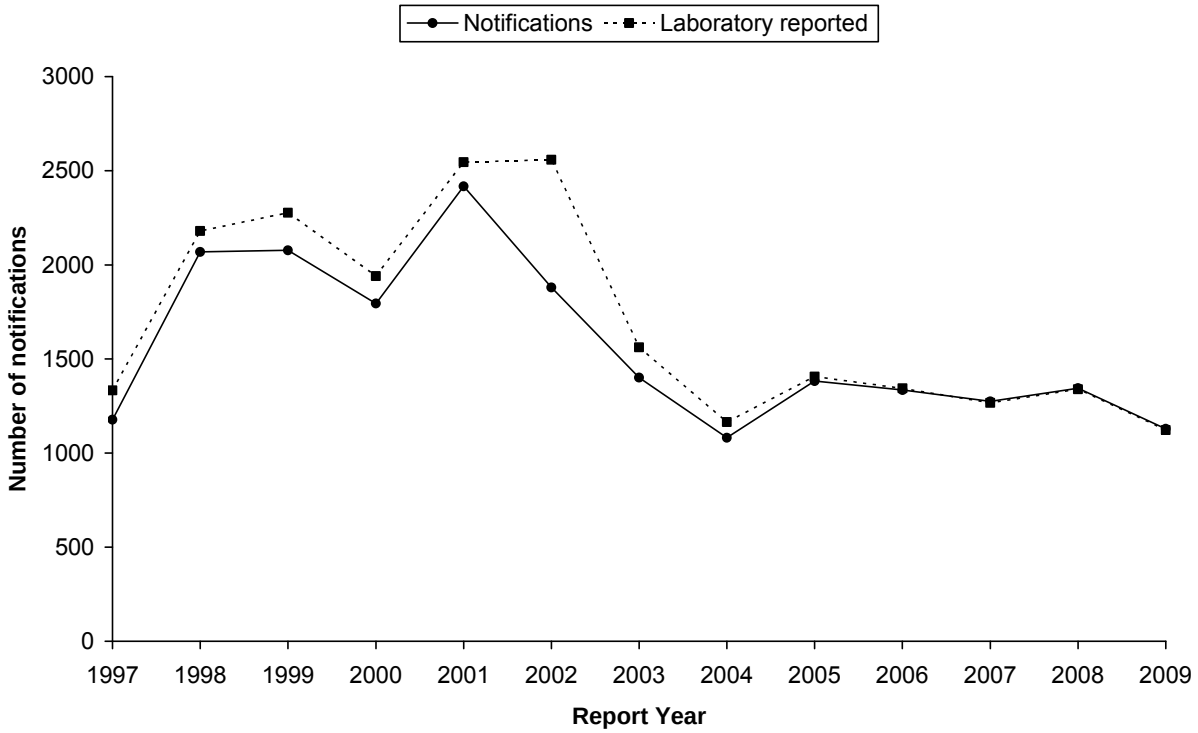
4.13.3 Notifiable disease data

4.13.3.1 Annual notification trend

From 1997 to 2001 there was a general annual increase in the number of salmonellosis notifications with the highest number reported in 2001 (2 417 cases) (Figure 36). After 2001 the number of notifications decreased to a low in 2004 (1 081 cases), and has remained stable at 1 129 to 1 382 notifications per year since. The number of notifications in 2009 was the lowest since 2004.

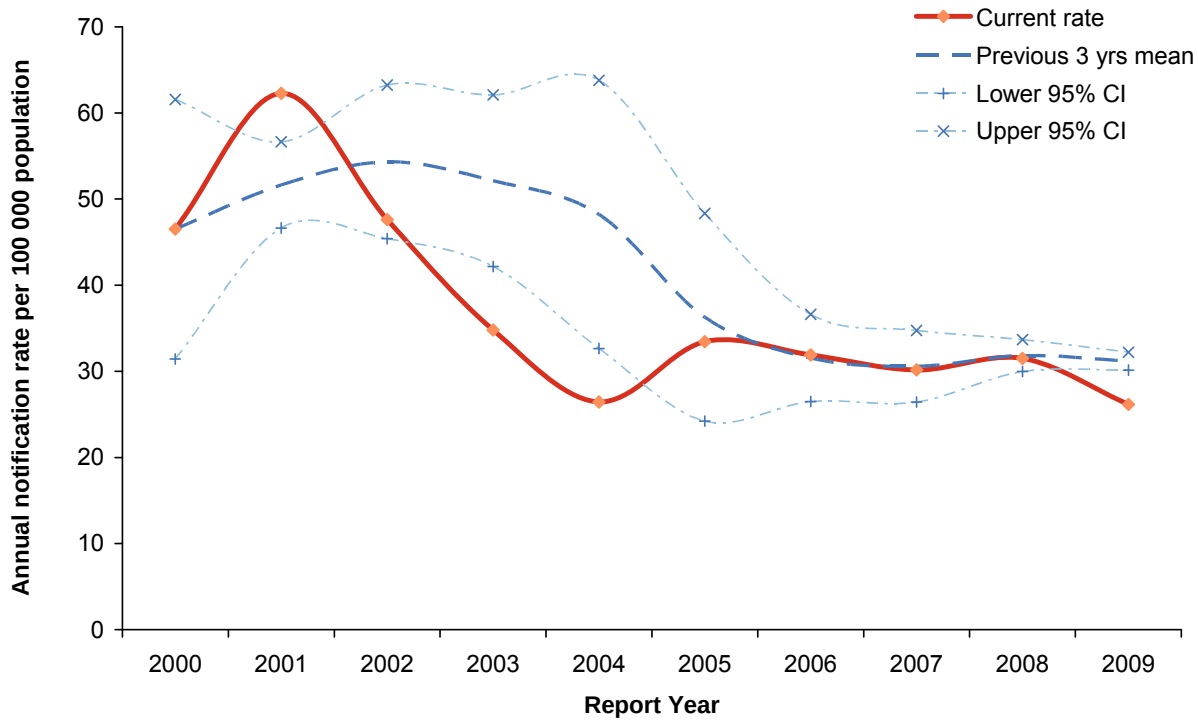
The change to direct laboratory notifications suggests that any differences between the number of notified cases and the number of laboratory reported cases should disappear in the future. Data for 2008 and 2009 support this conclusion.

Figure 36: Salmonellosis notifications and laboratory reported cases by year, 1997-2009



The 2009 salmonellosis notification rate was 26.2 per 100 000 population. Over the 10 year period from 2000 to 2009 the salmonellosis annual notification rate was highest in 2001 before decreasing from 2002 to 2004 and levelling off after that (Figure 37).

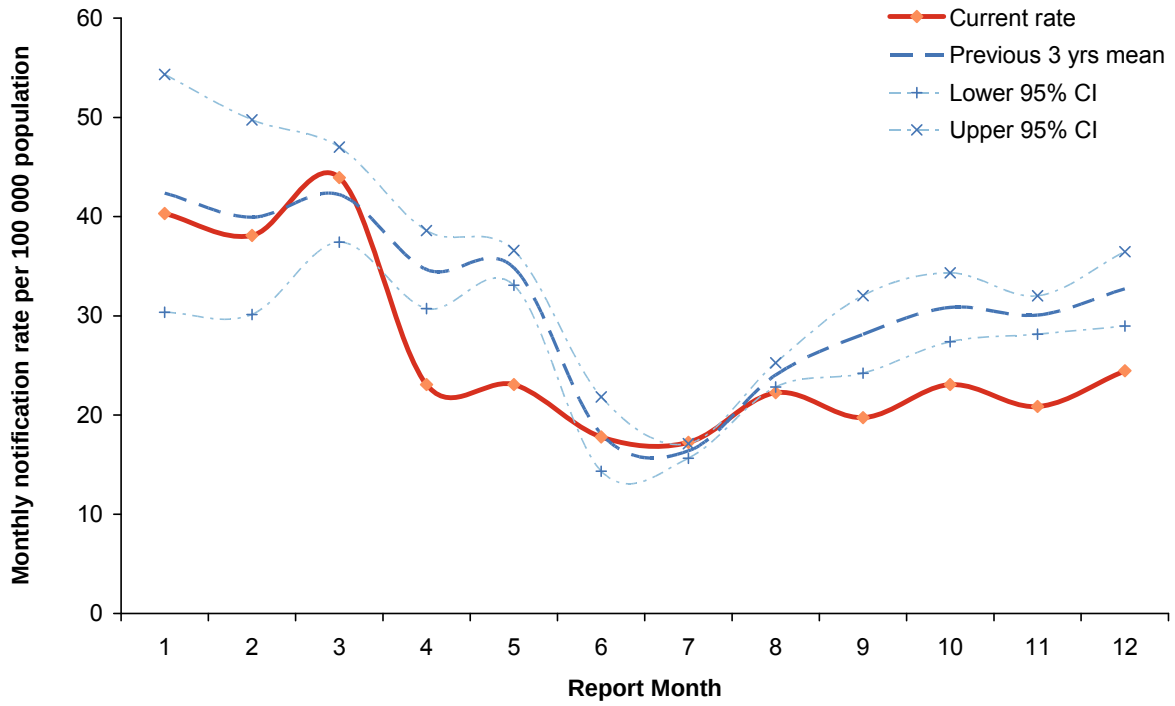
Figure 37: Salmonellosis notification rate by year, 2000-2009



4.13.3.2 Seasonality

Salmonellosis notifications reported per 100 000 population by month for 2009 show a clear seasonal pattern with notifications being highest during summer and autumn and lowest in mid-winter (Figure 38). A similar trend is seen in the historic mean rate.

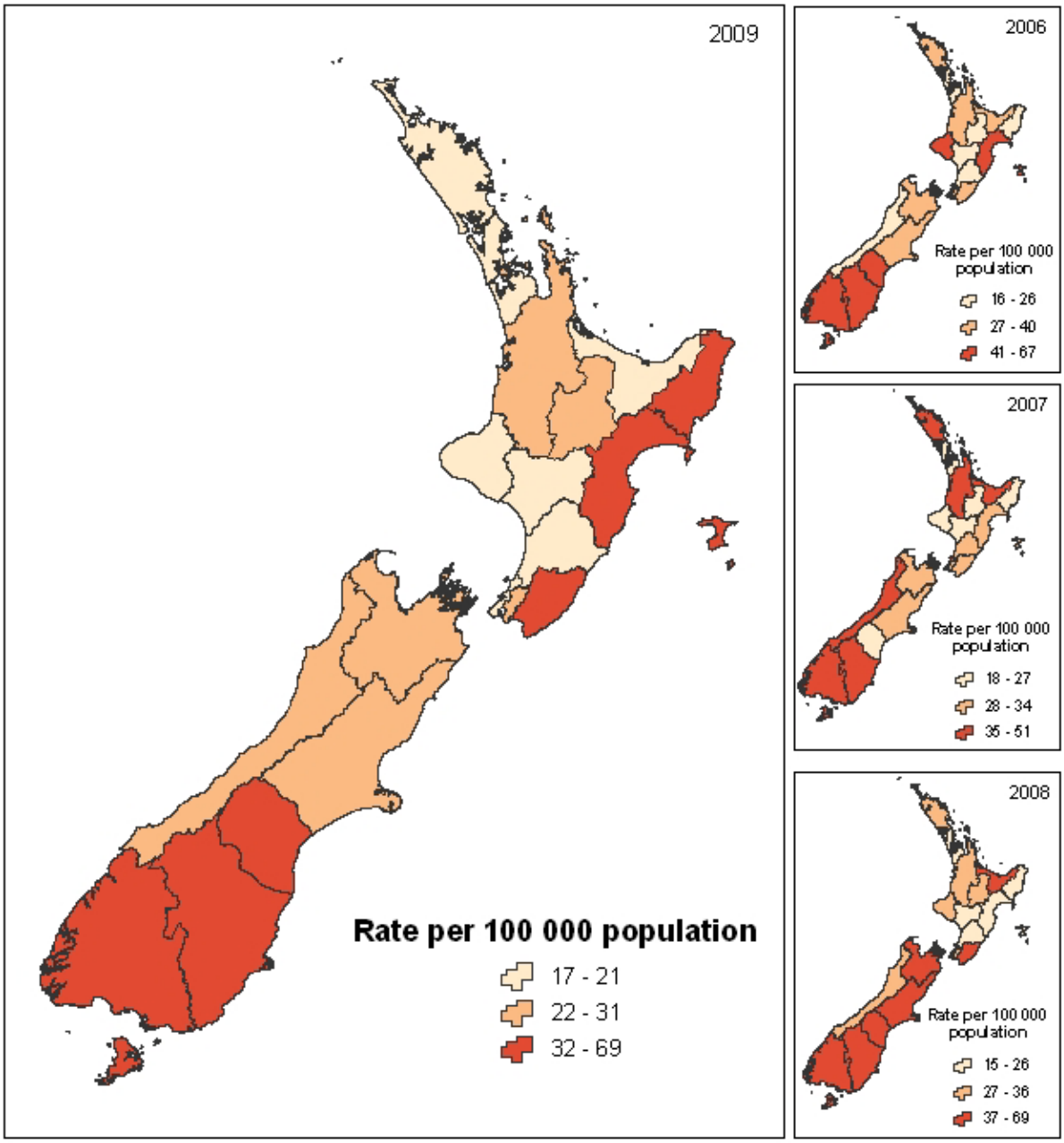
Figure 38: Salmonellosis notification monthly rate (annualised) for 2009



4.13.3.3 Geographic distribution of salmonellosis notifications

Rates of salmonellosis vary throughout the country as illustrated in Figure 39. The highest salmonellosis notification rate in 2008 was reported in Tairawhiti DHB (69.3 per 100 000 population, 32 cases), followed by South Canterbury (61.2 per 100 000, 34 cases) and Southland (50.1 per 100 000, 56 cases) DHBs. Otago and Southland DHBs have consistently featured in the highest quantile of salmonellosis notification rates for each of the last four years.

Figure 39: Geographic distribution of salmonellosis notifications, 2006-2009



4.13.3.4 Age and sex distribution of salmonellosis cases

In 2009, the numbers and rates of notifications and hospitalisations for salmonellosis were generally similar for males and females. There were slightly more hospitalisations for females compared to males (Table 45).

Table 45: Salmonellosis cases by sex, 2009

Sex	EpiSurv notifications		Morbidity[a]		Deaths recorded in EpiSurv
	No.	Rate[b]	No.	Rate[b]	No.
Male	555	26.2	72	3.4	
Female	564	25.7	86	3.9	1
Unknown	10				
Total	1 129	26.2	158	3.7	1

[a] MoH morbidity data for hospital admissions
[b] per 100 000 of population

In 2009, age-specific salmonellosis rates were highest for those aged less than 1 year for both the notifications (123.7 per 100 000 population, 78 cases) and hospitalisations (34.9 per 100 000 population, 22 admissions) (Table 46). Those in the 1 to 4 years age group also reported high salmonellosis notification and hospitalisation rates compared to other age groups.

Table 46: Salmonellosis cases by age group, 2009

Age group	EpiSurv notifications		Morbidity[a]		Deaths recorded in EpiSurv
	No.	Rate[b]	No.	Rate[b]	No.
<1	78	123.7	22	34.9	1
1 to 4	218	89.9	16	6.6	
5 to 9	68	23.6	8	2.8	
10 to 14	38	12.8	3	-	
15 to 19	60	18.6	7	2.2	
20 to 29	144	24.6	22	3.8	
30 to 39	136	23.6	18	3.1	
40 to 49	121	19.1	17	2.7	
50 to 59	115	21.6	12	2.3	
60 to 69	72	18.3	18	4.6	
70+	75	19.7	15	3.9	
Unknown	4				
Total	1 129	26.2	158	3.7	1

[a] MoH morbidity data for hospital admissions
[b] per 100 000 of population. Where fewer than five cases have been reported a rate has not been calculated.

4.13.3.5 Risk factors reported

The most commonly reported risk factors for salmonellosis notified cases during 2009 were consumption of food from retail premises (41.9%) followed by contact with farm animals (34.6%) and consumption of untreated water (28.5%) (Table 47).

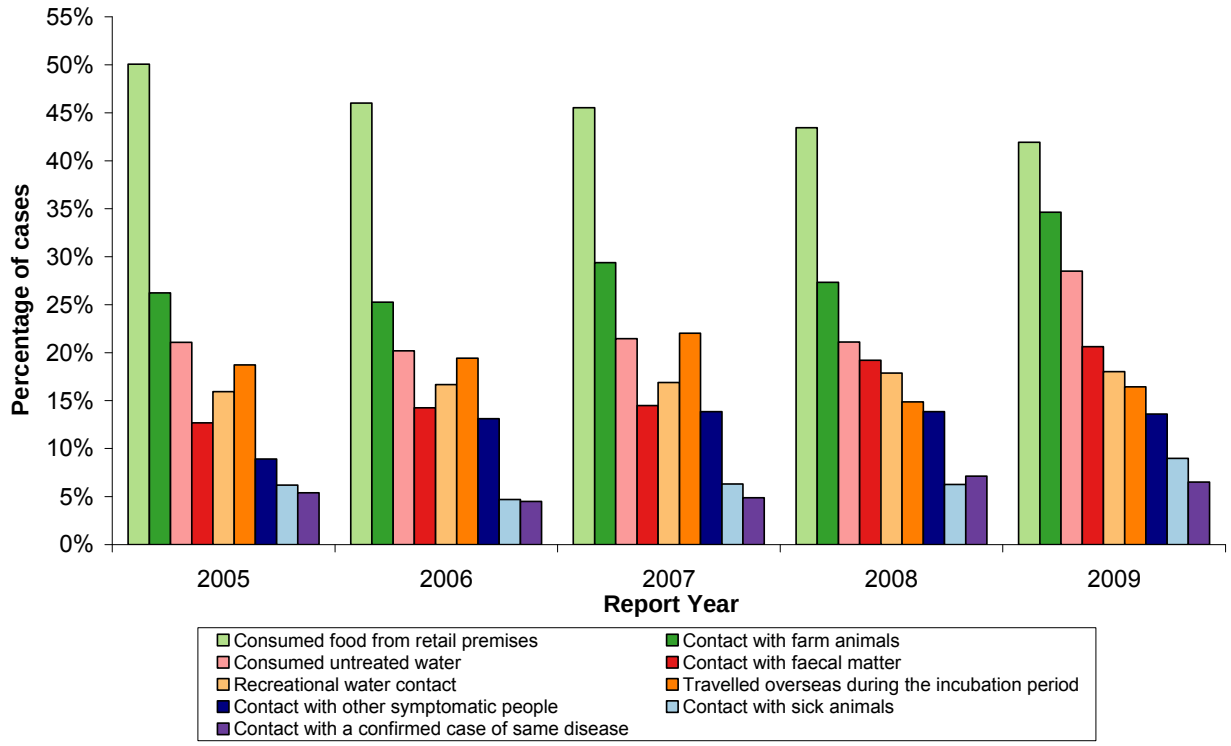
Table 47: Exposure to risk factors associated with salmonellosis, 2009

	Notifications			
Risk Factor	**Yes**	**No**	**Unknown**	**%[a]**
Consumed food from retail premises	200	277	652	41.9
Contact with farm animals	186	351	592	34.6
Consumed untreated water	116	291	722	28.5
Contact with faecal matter	94	362	673	20.6
Recreational water contact	88	400	641	18.0
Travelled overseas during the incubation period	98	498	533	16.4
Contact with other symptomatic people	66	419	644	13.6
Contact with sick animals	43	436	650	9.0

[a]Percentage refers to the cases that answered "yes" out of the total number of cases for which this information was supplied. Cases may have more than one risk factor recorded.

Between 2005 and 2009 the risk factors associated with salmonellosis cases have generally occurred in the same order of importance and to the same magnitude on a yearly basis (Figure 40). The most commonly reported risk factor for salmonellosis cases every year was consumption of food from retail premises, followed by contact with farm animals.

Figure 40: Salmonellosis risk factors by percentage of cases and year, 2005-2009



4.13.3.6 Estimate of travel-related cases

For cases where information on travel was provided, 16.4% (95%CI 13.6-19.7%) had travelled overseas during the incubation period. Assuming that the cases for which travel information was provided were representative of all salmonellosis cases, a Poisson distribution can be used to estimate the total number of potentially travel related cases of salmonellosis in 2009. The resultant distribution has a mean of 186 cases (95% CI 149-225).

If data from the last four years are considered the estimated proportion of cases travelling overseas within the incubation period of the organism is 18.4% (95% CI 17.0-19.8%).

4.13.4 Outbreaks reported as caused by *Salmonella* spp

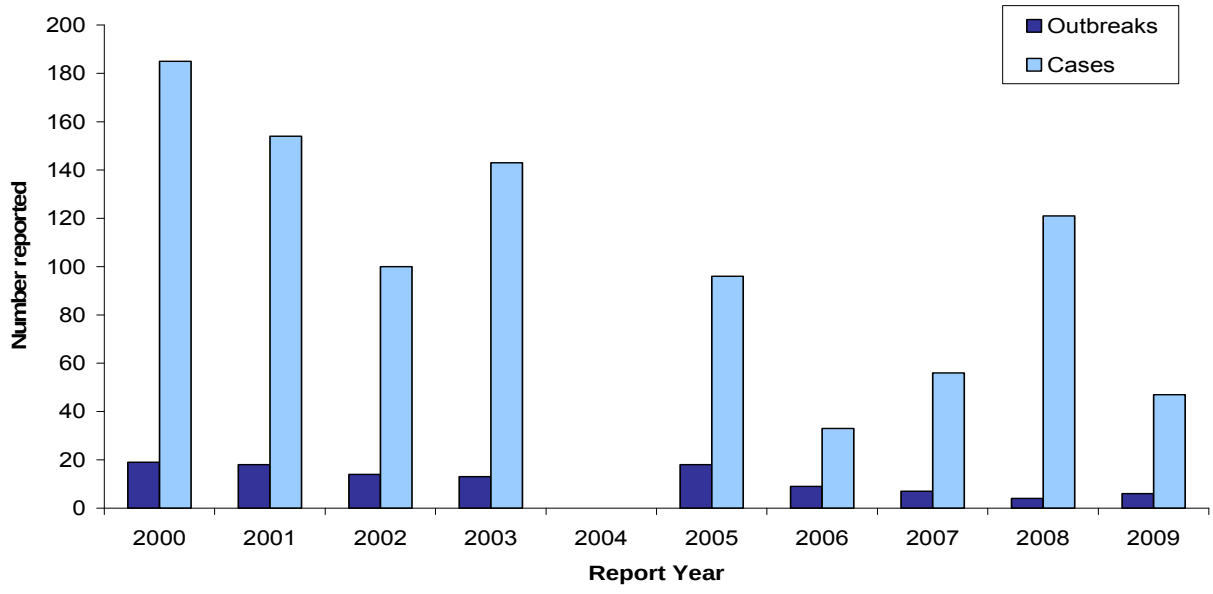
In 2009, there were 12 *Salmonella* spp. outbreaks reported and six of these were reported to be foodborne (Table 48). Nine of the 17 hospitalisations due to *Salmonella* spp. were associated with foodborne outbreaks.

Table 48: *Salmonella* spp. foodborne outbreaks reported, 2009

Measure (No.)	Foodborne *Salmonella* spp. outbreaks	All *Salmonella* spp. outbreaks
Outbreaks	6	12
Cases	47	76
Hospitalised cases	9	17

The number of foodborne outbreaks reported between 2000 and 2009 ranged from zero (2004) to 19 (2000), generally decreasing in number over time (Figure 41). The total number of cases associated with the outbreaks has also generally decreased over the period, although 2008 had the highest number of cases since 2003.

Figure 41: Foodborne *Salmonella* spp. outbreaks and associated cases reported by year, 2000–2009



4.13.4.1 Details of food-associated outbreaks

Table 49 contains details of the six food–associated *Salmonella* spp. outbreaks reported in 2009.

Table 49: Details of food-associated *Salmonella* spp. outbreaks, 2009

Public Health Unit (Month)	Suspected vehicle	Setting	Number ill	Confirmation
Auckland (January)	Unknown, possibly infected food handler	Restaurant/Café	10C	1, 2
Auckland (April)	Unknown	Overseas travel (Fiji)	1C, 1P	6
Canterbury (November)	Unknown	Airline	12C, 1P	7
Gisborne (February)	Watermelon	Home	15C	1, 3
Manawatu (August)	Farm environment, unpasteurised milk	Farm, Home	4C	1, 2, 5
Southland (April)	Unknown	Home	3C	2

C = confirmed, P = probable
Confirmation:
1 = Environmental investigation – identified critical control point failures linked to implicated source
2 = Epidemiological – case had history of exposure to implicated source
3 = Epidemiological – case control or cohort study showed elevated risk for cases to implicated source
4 = Laboratory – pathogen suspected to have caused illness identified in food handler
5 = Laboratory – pathogen suspected to have caused illness identified in implicated source (food)
6 = No evidence
7 = Other evidence

Evidence linking salmonellosis outbreaks to particular food vehicles was generally weak. However, the largest outbreak occurring in early 2009 included strong evidence for watermelon as the source of the outbreak.

4.13.4.2 Laboratory investigation of samples from suspected foodborne outbreaks

During investigations of suspected foodborne illness outbreaks by ESR's Public Health Laboratory, *Salmonella* spp. was detected in a faecal sample from one investigation, with shellfish implicated. However, norovirus was detected in the same faecal sample. *Salmonella* was detected in raw (unpasteurised) milk and environmental samples associated with another investigation.

4.13.5 *Salmonella* types commonly reported

4.13.5.1 Human isolates

A total of 1 122 non-typhoidal human isolates were confirmed and reported by ESR's Enteric Reference Laboratory during 2009. Of these isolates, 661 (58.9%) were *Salmonella* Typhimurium.

Table 50 shows the number of isolates of selected *Salmonella* types reported by the Enteric Reference Laboratory at ESR. The incidence of all *S.* Typhimurium definitive types (DT) has fluctuated between 2006 and 2009. DT160 remained the most common single type. However, the number of isolates of this type continues to decrease. The increase in typed isolates of *S.* Typhimurium DT1 from 2008 to 2009 is probably due to its involvement in significant outbreaks.

Table 50: Selected *Salmonella* serotypes and subtypes of laboratory-reported salmonellosis, 2006-2009

Subtype	2006	2007	2008	2009
S. Typhimurium	733	596	729	661
DT160	260	152	135	106
DT1	72	91	72	94
DT101	71	43	72	56
DT156	87	73	67	54
DT42	28	15	93	40
RDNC-May06	16	51	55	43
Other or unknown	199	171	235	268
S. Enteritidis	107	151	124	95
PT9a	53	60	45	39
PT1b	9	18	19	4
PT26	7	17	10	2
Other or unknown	38	56	50	50
S. Infantis	58	86	86	71
S. Brandenburg	55	47	33	36
S. Saintpaul	35	25	35	26
S. Mississippi	13	11	10	14
S. Virchow	13	34	14	12
S. Agona	24	13	10	10
Other or unknown serotypes	305	304	298	197
Total	**1 343**	**1 267**	**1 339**	**1 122**

4.13.5.2 Non-human isolates

A total of 888 non-human *Salmonella* isolates were typed by the Enteric Reference Laboratory during 2009 (Table 51).

Table 51: Selected *Salmonella* serotypes and subtypes from non-human sources, 2006-2009

Subtype	2006	2007	2008	2009	Major Sources, 2009
S. Typhimurium	543	333	727	388	
RDNC	33	52	104	67	Bovine (31), Poultry environmental (10)
DT101	189	73	146	48	Bovine (37)
DT1	40	36	63	42	Bovine (37)
DT9	27	11	34	32	Bovine (25), Ovine (6)
DT12a	22	8	39	32	Bovine (23)
DT156	27	24	55	31	Bovine (24)
DT160	75	30	47	26	Poultry feed (11)
Other or unknown	130	99	239	110	
S. Brandenburg	319	191	92	137	Ovine (70), Bovine (22), Avian (16), Food (9), Canine (6)
S. Hindmarsh	162	110	34	46	Ovine (36), Bovine (5)
S. Agona	34	22	26	36	Poultry environmental (22), Food (5)
S. Infantis	68	70	51	30	Meat and bone meal (9), Reptile (8), Environmental (7)
Other or unknown serotypes	291	275	419	251	
Total	**1 417**	**1 001**	**1 349**	**888**	

S. Brandenburg was the most commonly isolated serotype in non-human samples during 2009, with numbers increasing after a steady decline in recent years.

4.13.5.3 Outbreak types

Table 52 shows the number of hospitalised cases and total cases by subtype for foodborne *Salmonella* outbreaks reported during 2009. Two outbreaks were associated with *S.* Typhimurium phage type 1 and the remaining four outbreaks were associated with different subtypes. The largest outbreak, due to *S.* Typhimurium phage type 1 was associated with nine hospitalisations and 15 cases from the Gisborne region.

Table 52: *Salmonella* subtypes reported in foodborne outbreaks, 2009

Pathogen and Subtype	Outbreaks	Hospitalised cases	Total cases
Salmonella Typhimurium phage type 1	2	9	25
Salmonella Ferruch	1	0	2
Salmonella Typhimurium phage type 12a variant	1	0	13
Salmonella Typhimurium phage type 156	1	0	4
Salmonella Typhimurium phage type 42 variant	1	0	3

4.13.6 Recent surveys

A survey of microbiological hazards in conventional and organic fresh produce (n=891) isolated *Salmonella* from two domestic organic lettuce samples from the same grower (McIntyre and Cornelius, 2009). A site visit identified bird faeces as the likely source of the contamination.

4.13.7 Relevant New Zealand studies and publications

4.13.7.1 Reports

A systematic review of the aetiology of salmonellosis in New Zealand was carried out to examine the possible role of foodborne transmission and to identify the most relevant food vehicles (Wilson and Baker, 2009). The study concluded that contaminated food was very likely to be the cause of the majority (>50%) of salmonellosis cases, with poultry, pig meat and meat in general identified as very likely or likely to be moderate causes (10-30% of cases) of human salmonellosis.

Options for a comprehensive national *Salmonella* surveillance programme for New Zealand were considered (Lake and Sexton, 2009). It was concluded that there was value in the integration of human and non-human surveillance information. Achieving an integrated approach would involve systematic consultation and agreement between affected parties.

4.13.7.2 Journal papers

A Bayesian approach was used to estimate the contribution of various food sources to the human salmonellosis burden in New Zealand (Mullner *et al.*, 2009a). The majority of cases (60%) were attributed to pork, followed by poultry (21.2%) and beef and veal (11.5%). Eggs and lamb and mutton were estimated to be minor contributors to the salmonellosis burden (3.2 and 1.4% of cases, respectively). It was noted that data for *Salmonella* in pork were sparse and the attribution results should be interpreted with care.

Results of a survey of domestic (100 carcasses) and imported (110 carcasses) pork for *Salmonella* was published (Wong *et al.*, 2009). *Salmonella* was not isolated from domestic pork. The prevalence of *Salmonella* in imported pork was 3.6% (95% CI 1.0-9.0%). The survey sampling was conducted during an eight month period from October 2004 to May 2005.

4.13.8 Relevant regulatory developments

In March 2009, NZFSA released their Salmonella Risk Management Strategy 2009-2012[4]. The Strategy aims to achieve a 30% reduction in the reported annual incidence of foodborne salmonellosis after five years. The strategy focuses on non-typhoid *Salmonella* and begins with a primary focus on intelligence gathering from a wide range of food sectors.

The objectives of the *Salmonella* risk management strategy are to:

- Quantify the proportion of foodborne cases attributable to:
    - specific foods
    - animal feeds
    - domestically produced versus imported foods
    - multi-resistant and virulent *Salmonella* genotypes associated with foods

[4] http://www.nzfsa.govt.nz/foodborne-illness/salmonella/strategy/salmonella-risk-management-strategy-2009-012.pdf

- Identify sources of *Salmonella* contamination of specific foods and animal feeds
- Determine the relative value of different interventions throughout the food chain in reducing the risk of salmonellosis
- Make prioritised risk management decisions on appropriate *Salmonella* control measures across the food chain, and according to data availability
- Design and implement an effective monitoring and review programme to support strategic goals.

In May 2009, the National Microbiological Databases (NMD) Programme was amended to include a 12 month trial of testing of porcine carcasses at primary processing, which commenced in October 2009[5].

4.14 Shigellosis

Summary data for shigellosis in 2009 are given in Table 53.

Table 53: Summary surveillance data for shigellosis, 2009

Parameter	Value in 2009	Section reference
Number of cases	119	4.14.2
Rate (per 100,000)	2.8	4.14.2
Hospitalisations (%)	19 (16.0%)	4.14.2
Deaths (%)	0 (0.0%)	4.14.2
Estimated travel-related cases (%)	77 (64.9%)	4.14.3.6
Estimated food-related cases (%)	NA	

NA = not applicable, no information is available on the food attributable proportion of shigellosis in New Zealand

4.14.1 Case definition

Clinical description: Shigellosis presents as gastroenteritis

Laboratory test for diagnosis: Isolation of *Shigella* spp. from a clinical specimen

Case classification:

Probable: A clinically compatible illness that is either a contact of a confirmed case of the same disease, or has had contact with the same common source i.e., is part of an identified common source outbreak

Confirmed: A clinically compatible illness that is laboratory confirmed

4.14.2 Shigellosis cases reported in 2009 by data source

During 2009, 119 notifications (2.8 cases per 100 000 population) of shigellosis and no resulting deaths were reported in EpiSurv. The Enteric Reference Laboratory at ESR confirmed and reported 114 *Shigella* isolates (2.6 per 100 000 population).

[5] http://www.nzfsa.govt.nz/animalproducts/legislation/notices/animal-material-product/nmd/nmd-09-schedule-1-technical-procedures.pdf

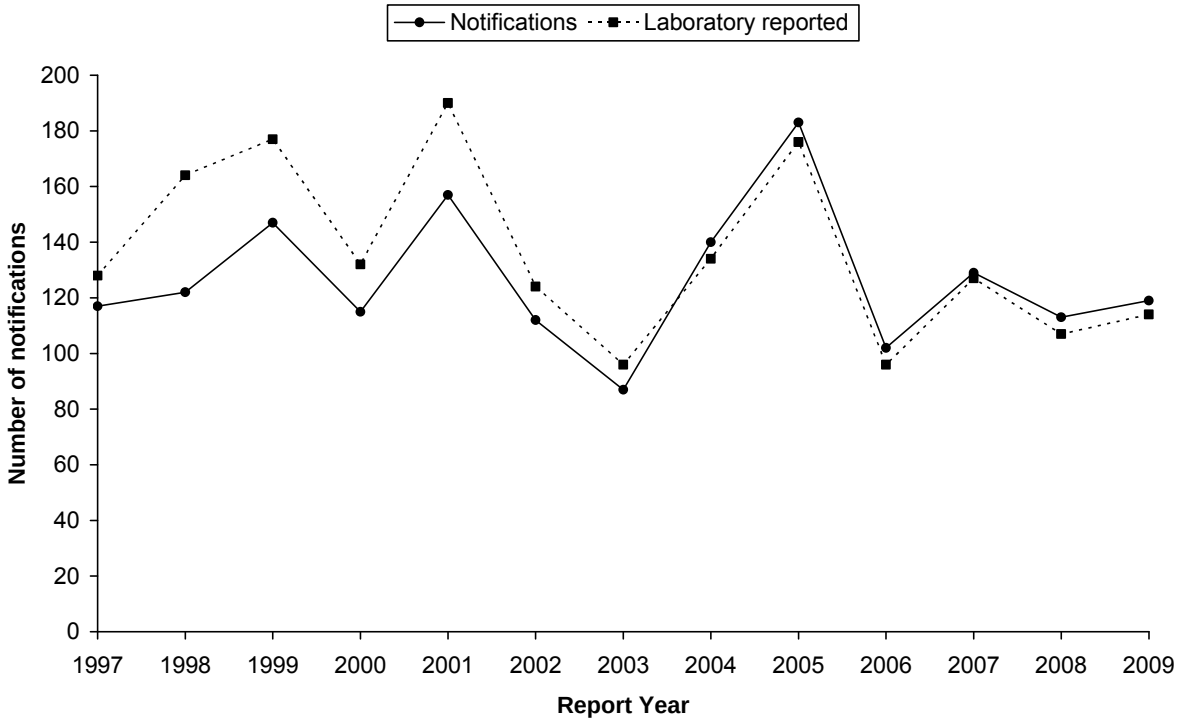
The ICD-10 code A03 was used to extract shigellosis hospitalisation data from the MoH NMDS database. Of the 19 hospital admissions (0.4 admissions per 100 000 population) recorded in 2009, 14 were reported with shigellosis as the primary diagnosis and five with shigellosis as another relevant diagnosis.

4.14.3 Notifiable disease data

4.14.3.1 Annual notification trend

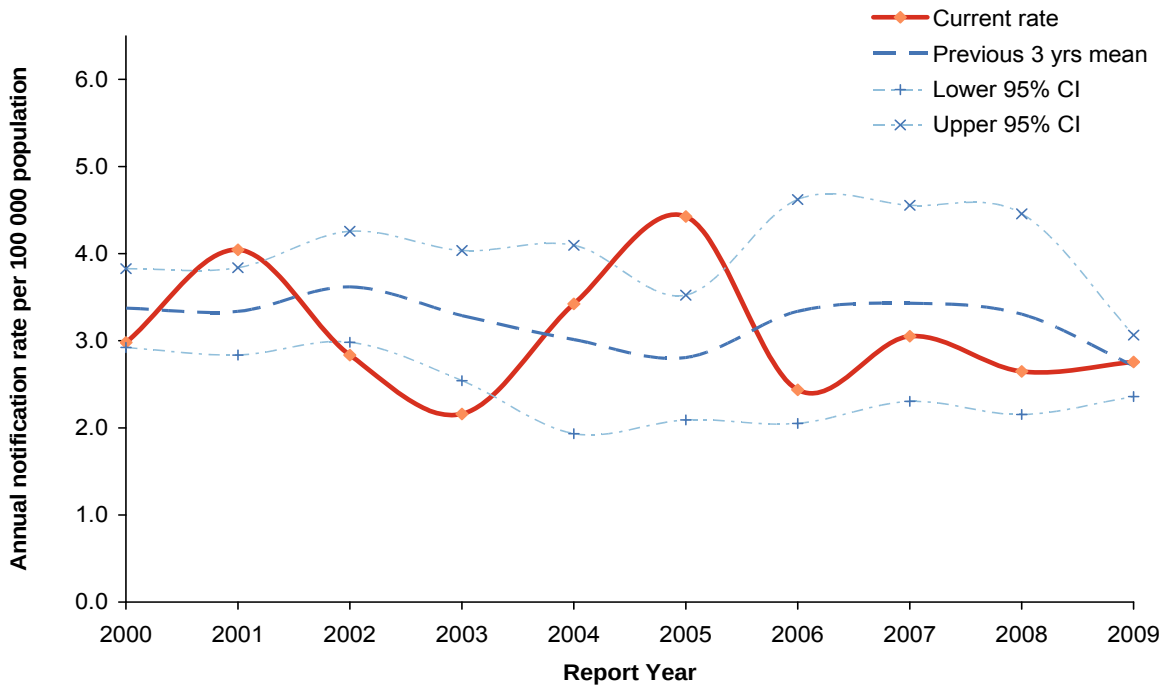
The number of notifications and laboratory reported cases of shigellosis fluctuates from year to year, but without any clear pattern (Figure 42). Numbers of notifications have been very stable during the period 2006-2009.

Figure 42: Shigellosis notifications and laboratory reported cases by year, 1997-2009



The 2009 shigellosis notification rate was 2.8 per 100 000 population. Between 2000 and 2006, the shigellosis annual notification rate fluctuated and was lowest in 2003 (2.2 per 100 000) and highest in 2005 (4.4 per 100 000). Since 2007 the annual notification rates have been levelling off (Figure 43).

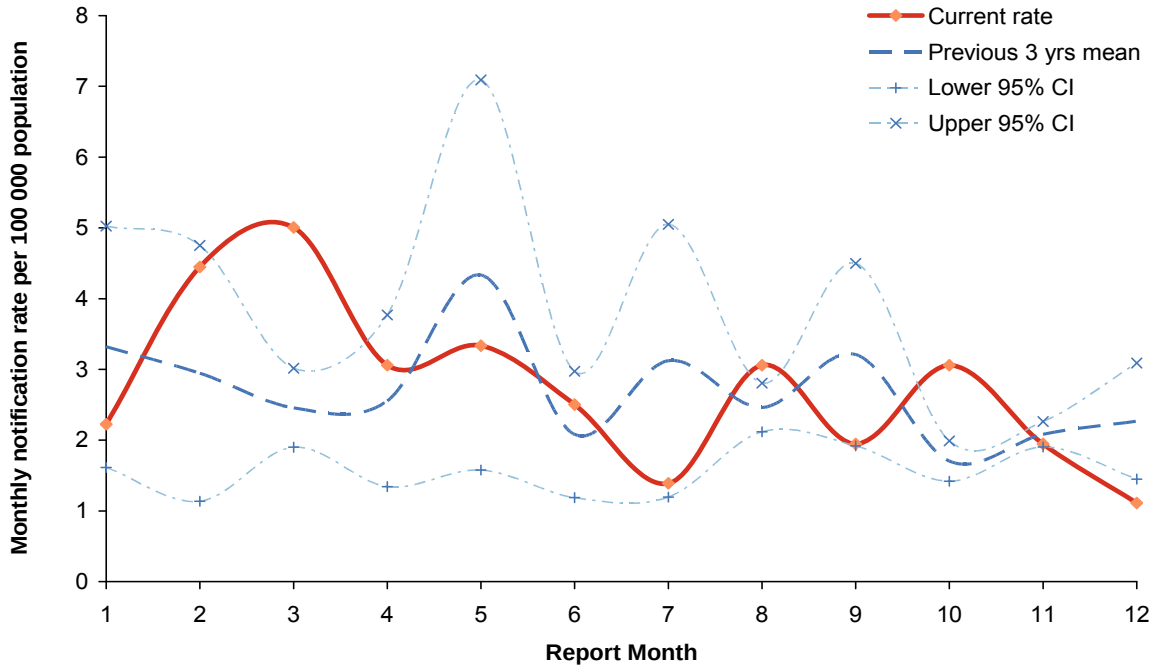
Figure 43: Shigellosis notification rate by year, 2000-2009



4.14.3.2 Seasonality

The number of notified cases of shigellosis per 100 000 population by month for 2009 is shown in Figure 44. In 2009, the shigellosis notification rate was highest in March and lowest in December.

Figure 44: Shigellosis monthly rate (annualised) for 2009



4.14.3.3 Age and sex distribution of shigellosis cases

In 2009, the numbers and rates of notifications and hospitalisations for shigellosis were similar for males and females (Table 54).

Table 54: Shigellosis cases by sex, 2009

Sex	EpiSurv notifications		Morbidity[a]		Deaths recorded in EpiSurv
	No.	Rate[b]	No.	Rate[b]	No.
Male	57	2.7	8	0.4	
Female	61	2.8	11	0.5	
Unknown	1				
Total	119	2.8	19	0.4	

[a] MoH morbidity data for hospital admissions
[b] per 100 000 of population

Age-specific shigellosis notification rates were highest for those in the 1 to 4 years and the 20 to 29 years age groups. The hospitalisation rates were not defined for most age groups due to the small number of cases. (Table 55).

Table 55: Shigellosis cases by age group, 2009

Age group	EpiSurv notifications		Morbidity[a]		Deaths recorded in EpiSurv
	No.	Rate[b]	No.	Rate[b]	No.
<1	2	-	0	-	
1 to 4	11	4.5	2	-	
5 to 9	8	2.8	4	-	
10 to 14	4	-	0	-	
15 to 19	4	-	0	-	
20 to 29	25	4.3	2	-	
30 to 39	17	2.9	0	-	
40 to 49	18	2.8	3	-	
50 to 59	12	2.3	2	-	
60 to 69	12	3.1	5	1.3	
70+	6	1.6	1	-	
Unknown	0				
Total	119	2.8	19	0.4	

[a] MoH morbidity data for hospital admissions
[b] per 100 000 of population. Where fewer than five cases have been reported a rate has not been calculated.

4.14.3.4 Risk factors reported

The most commonly reported risk factor for shigellosis in 2009 was overseas travel during the incubation period, followed by consumption of food from retail premises and recreational water contact (Table 56).

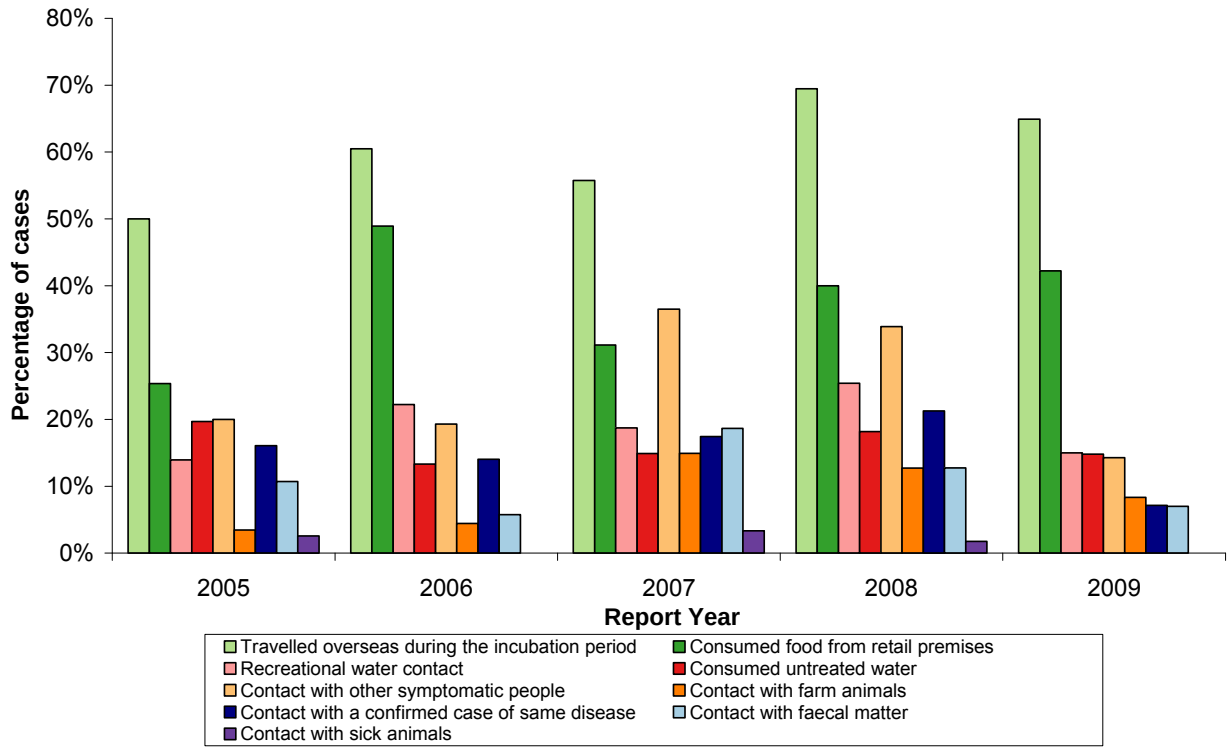
Table 56: Exposure to risk factors associated with shigellosis, 2009

	Notifications			
Risk Factor	**Yes**	**No**	**Unknown**	**%[a]**
Travelled overseas during the incubation period	50	27	42	64.9
Consumed food from retail premises	19	26	74	42.2
Recreational water contact	6	34	79	15.0
Consumed untreated water	4	23	92	14.8
Contact with other symptomatic people	6	36	77	14.3
Contact with farm animals	4	44	71	8.3
Contact with a confirmed case of same disease	2	26	91	7.1
Contact with faecal matter	3	40	76	7.0
Contact with sick animals	0	38	81	0.0

[a]Percentage refers to the cases that answered "yes" out of the total number of cases for which this information was Supplied. Cases may have more than one risk factor recorded.

Between 2005 and 2009, overseas travel during the incubation period and consumption of food from retail premises were the two most commonly reported risk factors for shigellosis each year (Figure 45).

Figure 45: Shigellosis risk factors by percentage of cases and year, 2005-2009



4.14.3.5 Estimate of travel-related cases

For cases where information on travel was provided, 64.9% (95%CI 53.2-75.5%) had travelled overseas during the incubation period. Assuming that the cases for which travel information was provided were representative of all shigellosis cases, a Poisson distribution can be used to estimate the total number of potentially travel related cases of shigellosis in 2009. The resultant distribution has a mean of 77 cases (95% CI 55-101).

If data from the last four years are considered the estimated proportion of cases travelling overseas within the incubation period of the organism is 63.6% (95% CI 58.1-68.7%).

4.14.4 Outbreaks reported as caused by *Shigella* spp.

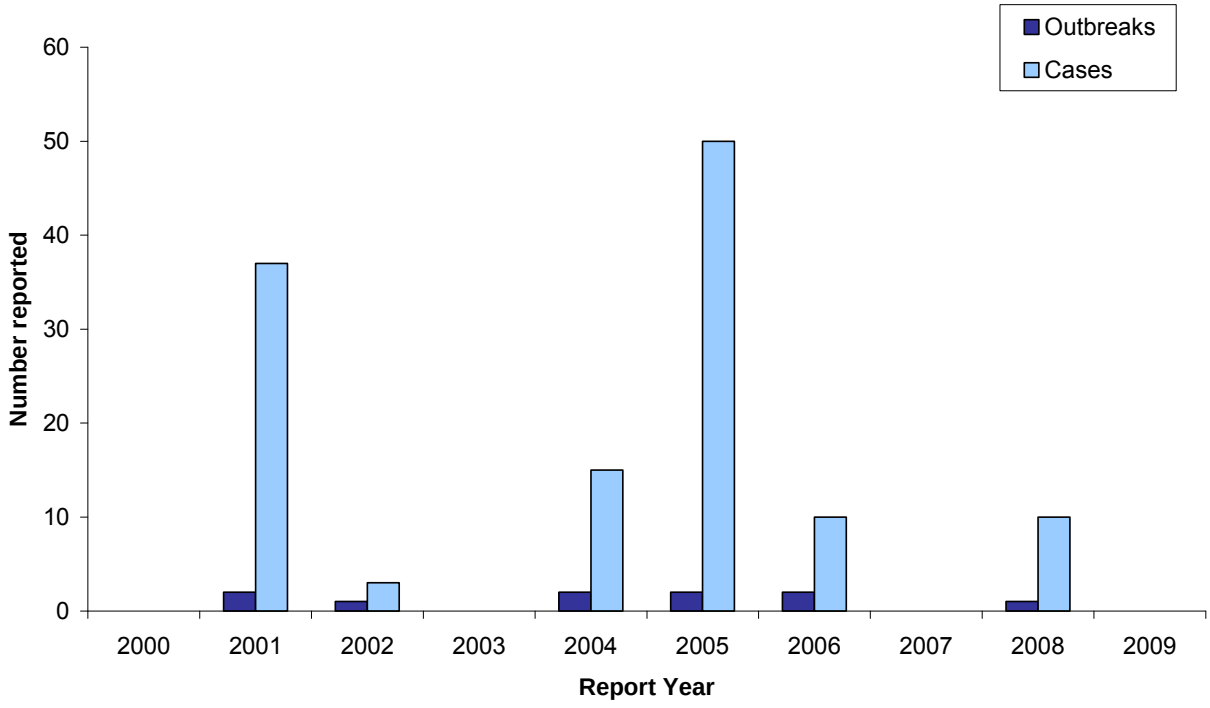
No foodborne *Shigella* outbreaks were reported in 2009 (Table 57).

Table 57: *Shigella* spp. outbreaks reported, 2009

Measure (No.)	Foodborne *Shigella* spp. outbreaks	All *Shigella* spp. outbreaks
Outbreaks	0	3
Cases	0	8
Hospitalised cases	0	0

Foodborne shigellosis outbreaks are rare with not more than two outbreaks being reported each year from 2000 to 2009 (Figure 46).

Figure 46: *Shigella* outbreaks and associated cases reported by year, 2000-2009



4.14.4.1 Details of food-associated outbreaks

No foodborne *Shigella* outbreaks were reported in 2009

4.14.4.2 Laboratory investigation of samples from suspected foodborne outbreaks

During investigations of suspected foodborne illness outbreaks by ESR's Public Health Laboratory, no samples were found to contain *Shigella* spp.

4.14.5 *Shigella* types commonly reported

There were 114 isolates of *Shigella* spp. confirmed and reported by the Enteric Reference Laboratory at ESR in 2009, compared with 107 in 2007. The species and major serogroups identified in 2009 were distributed as follows: *S. sonnei* biotypes (64.0%, 73 isolates, including 36 Biotype g and 33 Biotype a), *S. flexneri* (27.2%, 31 isolates, including 13 type 2a and 6 type 3a), *S.* boydii (7.0%, 8 isolates, including 3 type 4 and 2 type 1), and two isolates of *Shigella* species (1.8%).

4.14.6 Relevant New Zealand studies and publications

Nil.

4.14.7 Relevant regulatory developments

Nil.

4.15 *Staphylococcus aureus* Intoxication

4.15.1 Case definition

Clinical description:	Gastroenteritis with sudden severe nausea and vomiting
Laboratory test for diagnosis:	Detection of enterotoxin in faecal or vomit specimen or in leftover food or isolation of $\geq 10^3$/gram coagulase-positive *S. aureus* from faecal or vomit specimen or $\geq 10^5$ from leftover food
Case classification:	
Probable	A clinically compatible illness that is either a contact of a confirmed case of the same disease, or has had contact with the same common source i.e., is part of an identified common source outbreak
Confirmed	A clinically compatible illness that is laboratory confirmed

4.15.2 *Staphylococcus aureus* intoxication cases reported in 2009 by data source

During 2009, there was one notification of *Staphylococcus aureus* intoxication and no resulting deaths reported in EpiSurv.

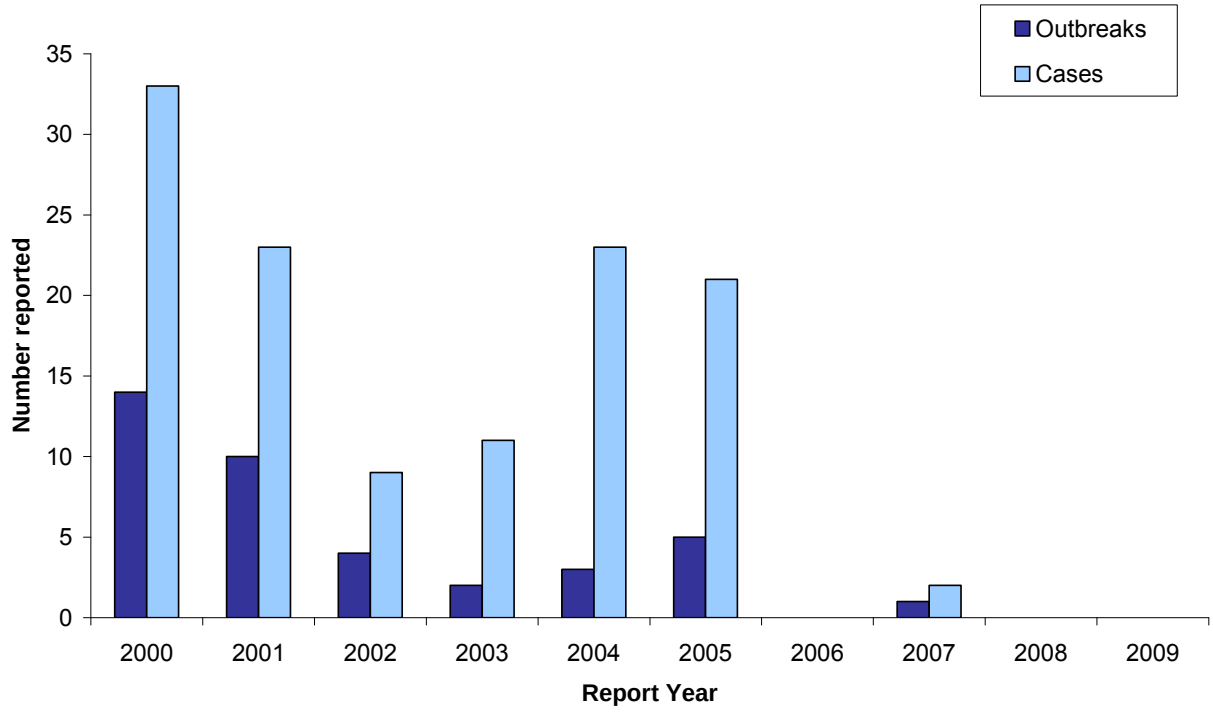
The ICD-10 code A05.0 was used to extract foodborne staphylococcal intoxication hospitalisation data from the MoH NMDS database. Of the four hospital admissions recorded in 2009, all were reported with foodborne staphylococcal intoxication as the primary diagnosis.

4.15.3 Outbreaks reported as caused by *Staphylococcus aureus*

In 2009, no *Staphylococcus aureus* outbreaks were reported in EpiSurv.

Between 2000 and 2003 there was a steady decrease in the number of *Staphylococcus aureus* outbreaks reported (Figure 47) followed by a small increase in 2004 and 2005. In 2006, 2008 and 2009, no *Staphylococcus aureus* outbreaks were reported in EpiSurv.

Figure 47: Foodborne *Staphylococcus aureus* outbreaks and associated cases reported by year, 2000 – 2009



4.15.3.1 Details of food-associated outbreaks

In 2009, no *Staphylococcus aureus* outbreaks were reported in EpiSurv.

4.15.3.2 Laboratory investigation of samples from suspected foodborne outbreaks

During investigations of suspected foodborne illness outbreaks by ESR's Public Health Laboratory, no samples were found to contain *S. aureus* or its enterotoxin.

4.15.4 Relevant New Zealand studies and publications

NZFSA published an article in Food Focus entitled "Hard to see but a nasty bite – Staphylococcus", which outlined sources, growth and survival characteristics, symptoms of illness and prevention strategies for *Staphylococcus aureus* and its intoxication:
http://www.nzfsa.govt.nz/publications/food-focus/2009-02/page-18.htm

4.15.5 Relevant regulatory developments

Nil.

4.16 Toxic Shellfish Poisoning

4.16.1 Case definition

Due to the diverse nature of toxins that may cause toxic shellfish poisoning, no consistent clinical description is provided for this condition. Depending on the toxin involved toxic shellfish poisoning may results in various combinations of gastrointestinal, neurosensory, neurocerebellar/neuromotor, general neurological and other symptoms. Case definitions for suspected cases of toxic shellfish poisoning are:

Amnesic Shellfish Poisoning (ASP): Vomiting or diarrhoea or abdominal cramps occurring within 24 hours of consuming shellfish AND no other probable cause identified by microbiological examination of faecal specimen from the case or microbiological testing of leftover food AND/OR one or more of the neurological symptoms from group C (see below) occurring within 48 hours of consuming shellfish.

Diarrhoeic Shellfish Poisoning (DSP): Vomiting or diarrhoea occurring within 24 hours of consuming shellfish AND no other probable cause identified by microbiological examination of faecal specimen from the case or microbiological testing of leftover food.

Neurotoxic Shellfish Poisoning (NSP): Two or more of the neurological symptoms from groups A and B (see below) occurring within 24 hours of consuming shellfish.

Paralytic Shellfish Poisoning (PSP): Paraesthesia occurring within 12 hours of consuming shellfish AND one of the neurological symptoms from group B (see below).

Toxic Shellfish Poisoning (TSP) type unspecified: Vomiting or diarrhoea occurring within 24 hours of consuming shellfish AND no other probable cause identified by microbiological examination of faecal specimen from the case or microbiological testing of leftover food OR any of the neurological symptoms from groups A and B (see below) occurring within 24 hours of consuming shellfish OR one or more of the neurological signs/symptoms from group C (see below) occurring within 48 hours of consuming shellfish.

Case definitions for probable cases of toxic shellfish poisoning are:

Meets case definition for suspect case AND detection of relevant biotoxin at or above the regulatory limit in shellfish obtained from near or same site (not leftovers) within seven days of collection of shellfish consumed by case.
Current level:

ASP: 20 ppm domoic acid/100 g shellfish
DSP: 20 μg/100 g or 5 MU/100 g shellfish (MU = mouse units)
NSP: 20 MU/100 g shellfish
PSP: 80 μg/100 g shellfish

Case definitions for confirmed cases of toxic shellfish poisoning are:

Meets case definition for suspect case AND detection of TSP biotoxin in leftover shellfish at a level resulting in the case consuming a dose likely to cause illness.

Current dose level:
ASP: 0.05 mg/kg body weight
DSP: ingestion of 48 μg or 12 MU
NSP: 0.3 MU/kg body weight
PSP: 10 MU/kg body weight (≅ 2μg/kg body weight)

Clinical symptoms for assigning status:
Group A:
• paraesthesia - i.e. numbness or tingling around the mouth, face or extremities
• alteration of temperature sensation

Group B:
• weakness such as trouble rising from seat or bed
• difficulty swallowing
• difficulty breathing
• paralysis
• clumsiness
• unsteady walking
• dizziness/vertigo
• slurred/unclear speech
• double vision

Group C:
• confusion
• memory loss
• disorientation
• seizure
• coma

4.16.2 Toxic shellfish poisoning cases reported in 2009

During 2009, there were no notifications of toxic shellfish poisoning reported in EpiSurv.

The ICD-10 code T61.2 was used to extract hospitalisation data for 'other fish and shellfish poisoning' from the MoH NMDS database. There were no hospital admissions reported in 2009. Note that this ICD-10 code includes shellfish and other fish.

4.16.3 Outbreaks reported as caused by TSP

In 2009, there were no outbreaks due to toxic shellfish poisoning reported in EpiSurv.

4.17 VTEC/STEC Infection

Summary data for VTEC/STEC infection in 2009 are given in Table 58.

Table 58: Summary surveillance data for VTEC/STEC infection, 2009

Parameter	Value in 2009	Section reference
Number of cases	143	4.17.2
Rate (per 100,000)	3.3	4.17.2
Hospitalisations (%)	7 (4.9%)	4.17.2
Deaths (%)	1 (0.7%)	4.17.2
Estimated travel-related cases (%)	7 (5.2%)	4.17.3.5
Estimated food-related cases (%)*	54 (39.6%)	4.17.2

* For estimation of food-related cases it was assumed that the proportions derived from expert consultation would exclude travel-related cases

4.17.1 Case definition

Clinical description: An illness of variable severity characterised by diarrhoea (often bloody) and abdominal cramps. Illness may be complicated by haemolytic uraemic syndrome (HUS), or thrombotic thrombocytopaenic purpura (TTP)

Laboratory test for diagnosis: Isolation of Shiga toxin (verotoxin) producing *Escherichia coli* OR detection of the genes associated with the production of Shiga toxin in *E. coli*

Case classification:

Probable A clinically compatible illness that is either a contact of a confirmed case of the same disease, or has had contact with the same common source i.e., is part of an identified common source outbreak

Confirmed A clinically compatible illness that is laboratory confirmed

4.17.2 VTEC/STEC infection cases reported in 2009 by data source

During 2009, 143 notifications (3.3 cases per 100 000 population) of VTEC/STEC infection were reported in EpiSurv. The Enteric Reference Laboratory at ESR reported 145 confirmed isolates (3.4 per 100 000).

The ICD-10 code A043 was used to extract enterohaemorrhagic *Escherichia coli* infection hospitalisation data from the MoH NMDS database. Of the seven hospital admissions recorded in 2009, six were reported with enterohaemorrhagic *Escherichia coli* infection as the primary diagnosis and one with this condition as another relevant diagnosis.

One death due to VTEC/STEC (O157:H7) infection was recorded in EpiSurv in 2009. This is the first death due to VTEC/STEC reported in Episurv since 1998.

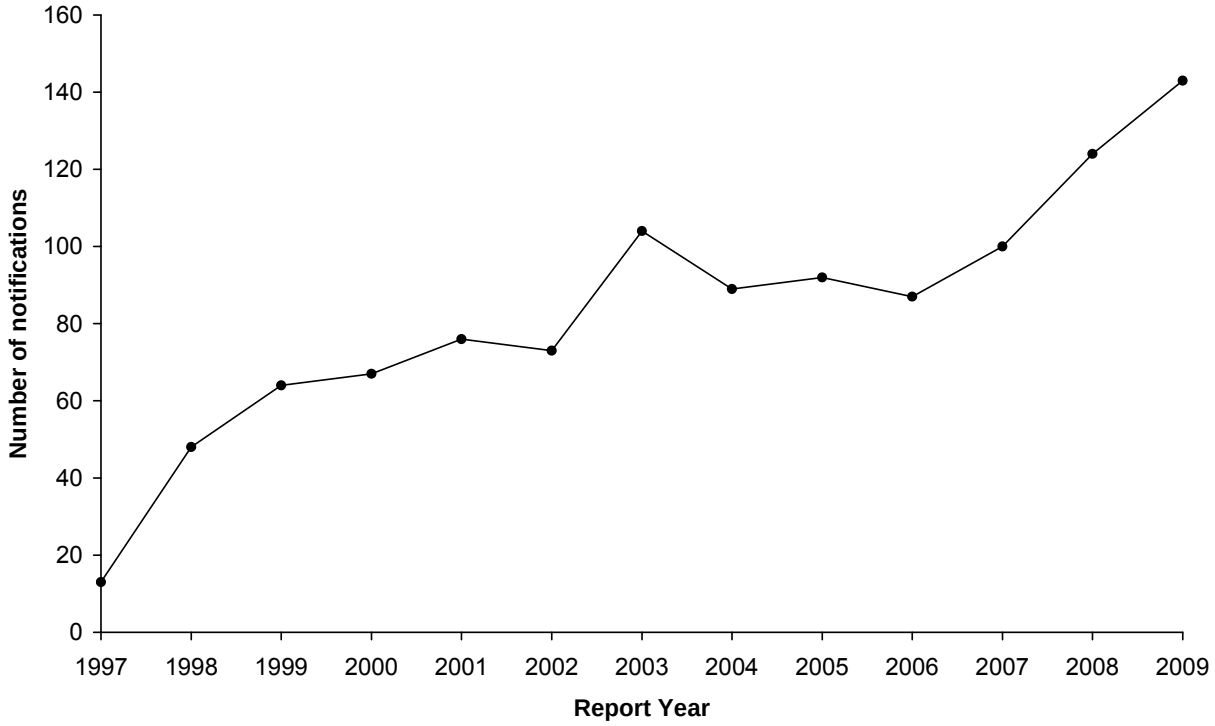
It has been estimated by expert consultation that 40% (minimum = 27%, maximum = 51%) of VTEC/STEC incidence is due to foodborne transmission. The expert consultation also estimated that approximately 30% of foodborne VTEC/STEC transmission was due to red meat of which two-thirds was considered to be due to consumption of uncooked, fermented, comminuted meat.

4.17.3 Notifiable disease data

4.17.3.1 Annual notification trend

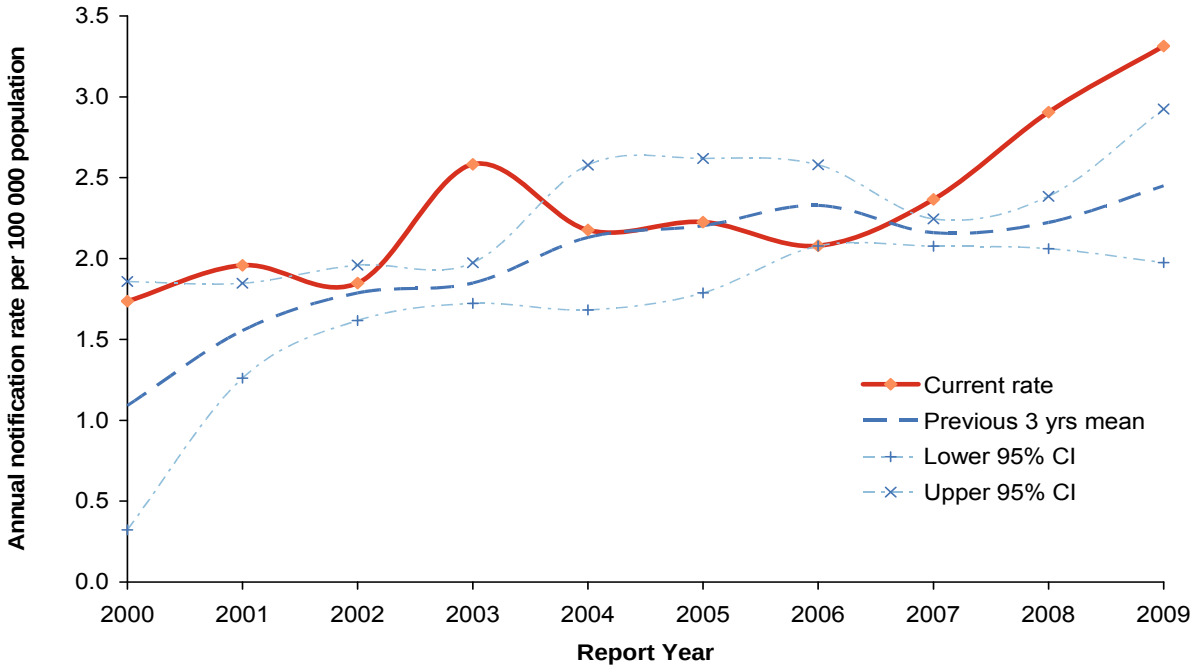
In 2009, 143 VTEC/STEC infection notifications were reported in EpiSurv. This is the highest number of notifications since VTEC/STEC became notifiable in 1996. There has been a general increase in the notifications of VTEC/STEC infection since 1997 (Figure 48).

Figure 48: VTEC/STEC infection notifications by year, 1997-2009



The 2009 VTEC/STEC infection notification rate was 3.3 per 100 000 population, the highest rate reported since 1996 (Figure 49). Between 2000 and 2009, the VTEC/STEC infection annual notification rate has shown a gradual increasing trend, with a slight peak in 2003.

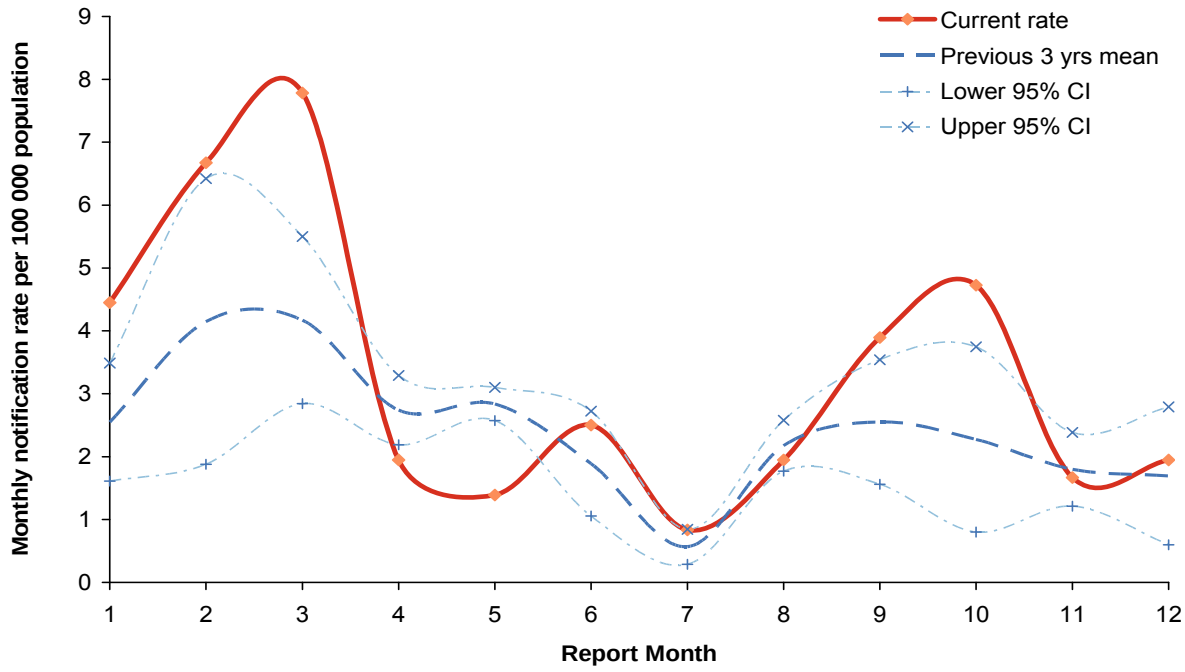
Figure 49: VTEC/STEC infection notification rate by year, 2000-2009



4.17.3.2 Seasonality

The number of notified cases of VTEC/STEC infection per 100 000 population by month for 2009 are shown in Figure 50. The 2009 monthly notification rate follows the historic mean rate trend with a peak in March and a trough in July. There was also a small peak in October 2009.

Figure 50: VTEC/STEC infection notification monthly rate (annualised) for 2009



4.17.3.3 Age and sex distribution of VTEC/STEC infection

In 2009, the sex-specific rate was higher in females than in males. The number of hospitalisations was similar for males and females (Table 59).

Table 59: VTEC/STEC infection by sex, 2009

Sex	EpiSurv notifications		Morbidity[a]		Deaths recorded in EpiSurv
	No.	Rate[b]	No.	Rate[b]	No.
Male	59	2.8	4	-	
Female	82	3.7	3	-	1
Unknown	2				
Total	143	3.3	7	0.2	1

[a] MoH morbidity data for hospital admissions
[b] per 100 000 of population. Where fewer than five cases have been reported a rate has not been calculated.

In 2009, the age specific VTEC/STEC infection notification rates were highest in the 1 to 4 years age group (21.9 per 100 000, 53 cases), followed by the less than one year age group (14.3 per 100 000, 9 cases). The 1 to 4 years age group had the highest number of hospitalisations (Table 60).

Table 60: VTEC/STEC infection by age group, 2009

Age group	EpiSurv notifications		Morbidity[a]		Deaths recorded in EpiSurv
	No.	Rate[b]	No.	Rate[b]	No.
<1	9	14.3	0	-	
1 to 4	53	21.9	3	-	
5 to 9	15	5.2	0	-	
10 to 14	3	-	0	-	
15 to 19	9	2.8	0	-	
20 to 29	12	2.1	2	-	
30 to 39	8	1.4	0	-	
40 to 49	7	1.1	0	-	
50 to 59	11	2.1	1	-	
60 to 69	9	2.3	0	-	
70+	7	1.8	1	-	1
Unknown	0				
Total	143	3.3	7	0.2	1

[a] MoH morbidity data for hospital admissions
[b] per 100 000 of population. Where fewer than five cases have been reported a rate has not been calculated.

4.17.3.4 Risk factors reported

In 2009, the most commonly reported risk factors for VTEC/STEC infection were contact with household pets (88.3%), consumption of dairy products (82.1%), consumption of raw fruit or vegetables (77.8%), and contact with farm animals (65.5%) (Table 61).

Table 61: Exposure to risk factors associated with VTEC/STEC infection, 2009

Risk Factor	Notifications			
	Yes	No	Unknown	%[a]
Contact with household pets	53	7	83	88.3
Consumed dairy products	64	14	65	82.1
Consumed raw fruit/vegetables	56	16	71	77.8
Contact with farm animals	36	19	88	65.5
Consumed poultry products	47	25	71	65.3
Consumed beef products	47	29	67	61.8
Consumed processed meats	36	36	71	50.0
Contact with animal manure	22	23	98	48.9
Consumed fruit/vegetables juice	28	37	78	43.1
Contact with children in nappies	25	57	61	30.5
Recreational water contact	26	66	51	28.3
Contact with other animals	12	32	99	27.3
Consumed home killed meats	20	56	67	26.3
Contact with persons with similar symptoms	19	70	54	21.3
Consumed lamb products	14	53	76	20.9
Consumed pink or undercooked meats	4	64	75	5.9
Consumed raw milk or products from raw milk	4	70	69	5.4
Travelled overseas during the incubation period	5	92	46	5.2

[a]Percentage refers to the cases that answered "yes" out of the total number of cases for which this information was supplied. Cases may have more than one risk factor recorded.

Between 2005 and 2009, the most consistently reported risk factors for VTEC/STEC infection were consumption of dairy products, consumption of raw fruit or vegetables (Figure 51), contact with household pets, and contact with farm animals (Figure 52).

Figure 51: VTEC/STEC infection foodborne risk factors by percentage of cases and year, 2005-2009

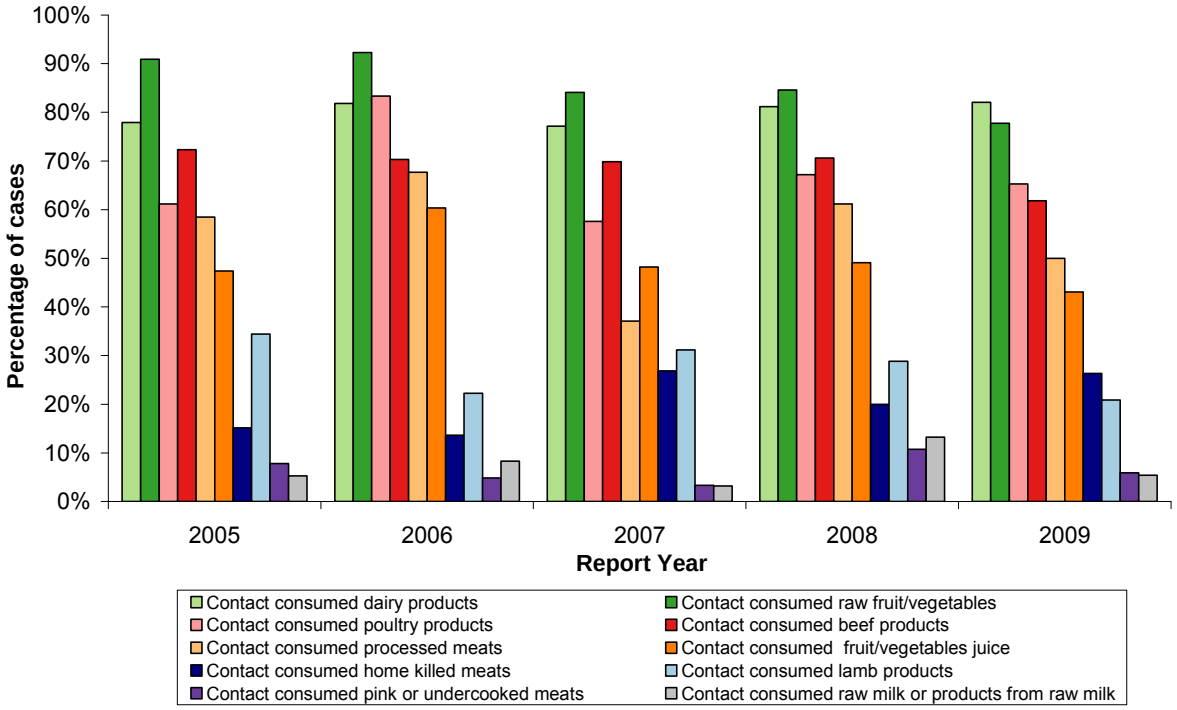
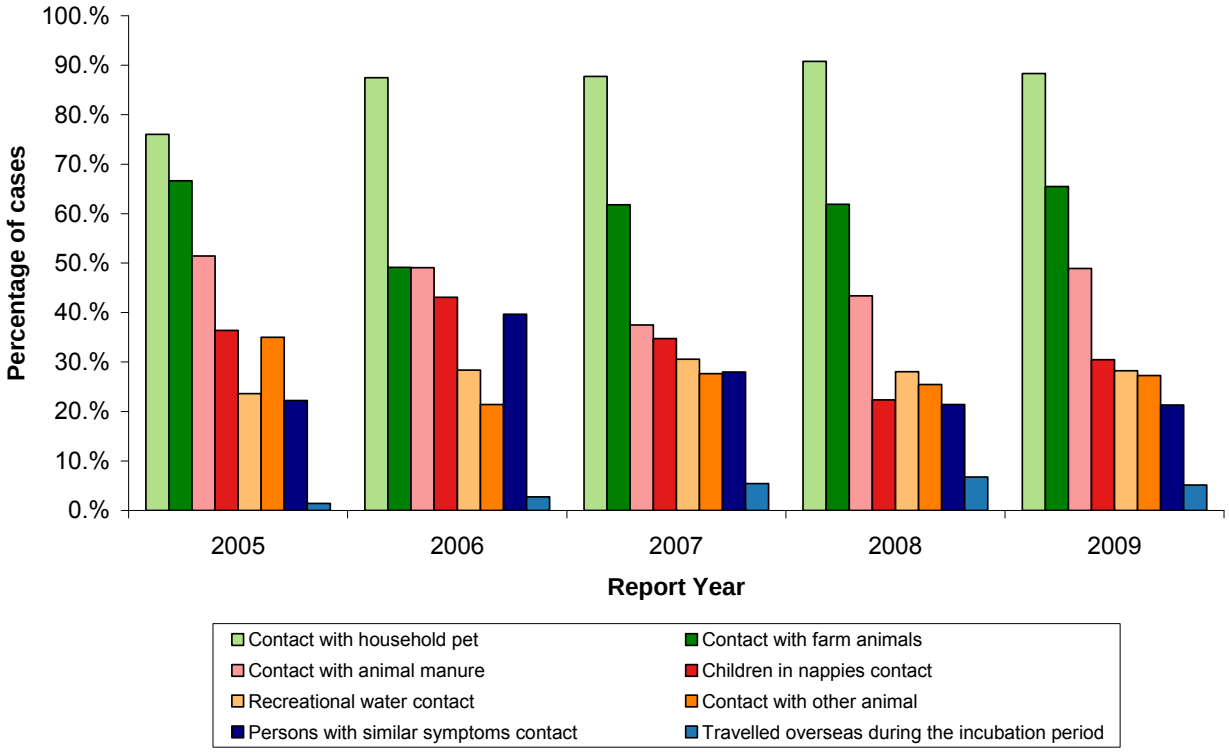


Figure 52: VTEC/STEC infection risk factors excluding food consumption by percentage of cases and year, 2005-2009



4.17.3.5 Estimate of travel-related cases

For cases where information on travel was provided, 5.2% (95%CI 1.7-11.6%) had travelled overseas during the incubation period. Assuming that the cases for which travel information was provided were representative of all VTEC/STEC infection cases, a Poisson distribution can be used to estimate the total number of potentially travel related cases of VTEC/STEC infection in 2008. The resultant distribution has a mean of 7 cases (95% CI 2-15).

4.17.4 Outbreaks reported as caused by VTEC/STEC

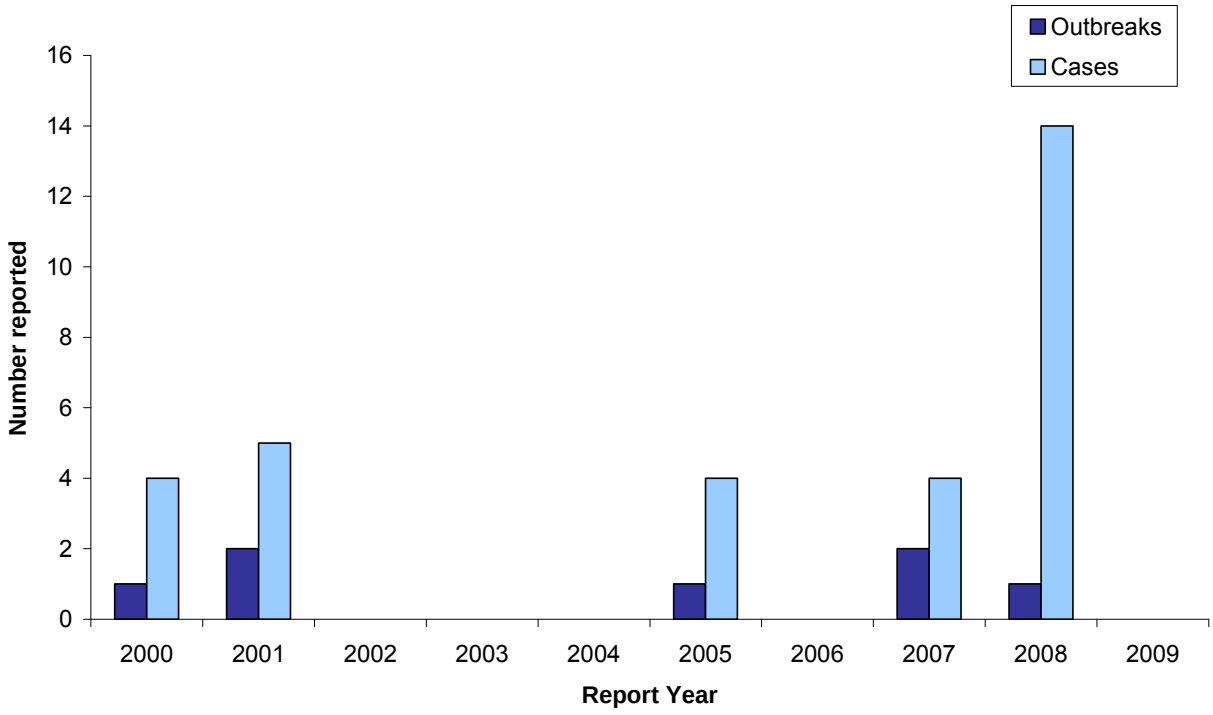
No foodborne VTEC/STEC outbreaks were reported in 2009 (Table 62).

Table 62: VTEC/STEC outbreaks reported, 2009

Measure (No.)	Foodborne VTEC/STEC outbreaks	All VTEC/STEC outbreaks
Outbreaks	0	4
Cases	0	15
Hospitalised cases	0	0

Over the ten year period from 2000 to 2009 there have been no more than two foodborne outbreaks of VTEC/STEC reported each year (Figure 53). Prior to 2008 there were no outbreaks reported that had more than five associated cases.

Figure 53: Foodborne VTEC/STEC outbreaks and associated cases reported by year, 2000–2009



4.17.4.1 Details of food-associated outbreaks

No foodborne VTEC/STEC outbreaks were reported in 2009.

4.17.4.2 Laboratory investigation of samples from suspected foodborne outbreaks

During investigations of suspected foodborne illness outbreaks by ESR's Public Health Laboratory, *E. coli* O157 was detected in venison mince implicated in a case of VTEC/STEC infection.

4.17.5 VTEC/STEC types commonly reported

A total of 145 VTEC/STEC isolates were received and typed in 2009. Of these, 137 (94.5%) were identified as serotype O157:H7, and eight as non-O157:H7. Of the eight non-O157:H7, three were typed as ONT:HNM, while the remaining five serotypes were all different. In comparison, 120 VTEC/STEC isolates (95 O157:H7 and two non-O157:H7) were received and typed in 2008.

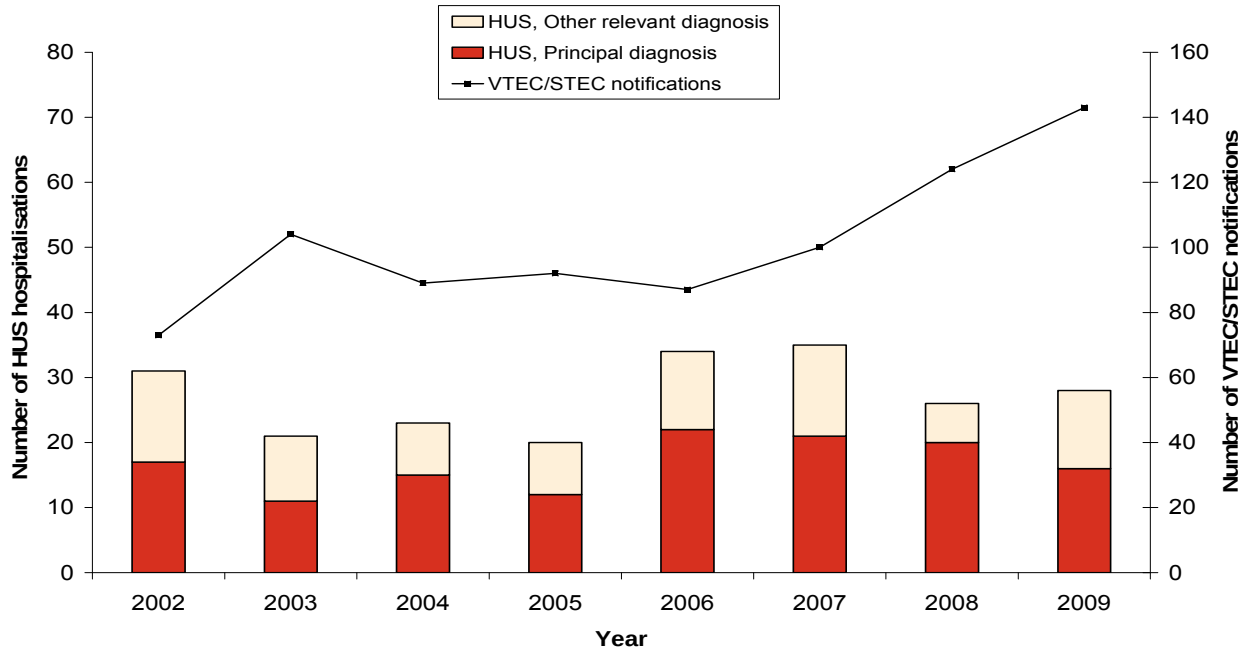
4.17.6 Disease Sequelae - haemolytic-uraemic syndrome (HUS)

HUS is a serious sequela of a VTEC/STEC enteric infection.

The ICD-10 code D59.3 was used to extract HUS hospitalisation data from the MoH NMDS database. Of the 28 hospitalised cases (0.6 admissions per 100 000 population) recorded in 2009, 16 were reported with HUS as the primary diagnosis and 12 with HUS as another relevant diagnosis.

Over the eight year period from 2002 to 2009, between 20 (in 2005) and 35 (in 2007) hospitalised cases for HUS have been reported each year. There is little evidence for a correlation between VTEC/STEC notifications and hospitalised HUS cases (Figure 54).

Figure 54: HUS hospitalised cases, 2002-2009



In 2009, the number of HUS hospitalised cases was greater for females than males (Table 63).

Table 63: HUS hospitalised cases by sex, 2009

Sex	Hospitalised cases[a]	
	No.	Rate[b]
Male	11	0.5
Female	17	0.8
Total	28	0.6

[a] M0H morbidity data for hospital admissions
[b] per 100 000 of population

In 2009 the highest age-specific hospitalised rate for HUS occurred for those aged less than five years (Table 64).

Table 64: HUS hospitalised cases by age group, 2009

Age groups	Hospitalised cases[a]	
	No.	Rate[b]
<5	6	2.0
5 to 9	4	-
10 to 14	0	-
15 to 19	5	1.5
20 to 29	0	-
30 to 39	4	-
40 to 49	2	-
50 to 59	3	-
60 to 69	2	-
70+	2	-
Total	28	0.6

[a] MoH morbidity data for hospital admissions
[b] per 100 000 of population. Where fewer than five cases have been reported a rate has not been calculated.

4.17.6.1 Haemolytic uraemic syndrome cases reported to the New Zealand Paediatric Surveillance Unit (NZPSU)

During 2009, five cases of HUS were reported to the NZPSU, with a mean age of 3.6 years (range 1.7 to 9.4 years). All five cases had a diarrhoeal prodrome. Four cases had *E. coli* O157:H7 isolated from their stools.

Source (Note: the details given above are from an advance excerpt from the NZPSU Annual Report, which had not been published at the time of finalisation of the current report. The source reference provided here is to the website where NZPSU Annual Reports are published): http://dnmeds.otago.ac.nz/departments/womens/paediatrics/research/nzpsu/annual_rpts.html

4.17.7 Recent surveys

Nil.

4.17.8 Relevant New Zealand studies and publications

Nil.

4.17.9 Relevant regulatory developments

Nil.

4.18 Yersiniosis

Summary data for yersiniosis in 2009 are given in Table 65.

Table 65: Summary surveillance data for yersiniosis, 2009

Parameter	Value in 2009	Section reference
Number of cases	431	4.18.2
Rate (per 100,000)	10.0	4.18.2
Hospitalisations (%)	46 (10.7%)	4.18.2
Deaths (%)	0 (0%)	4.18.2
Estimated travel-related cases (%)	30 (7.0%)	4.18.3.6
Estimated food-related cases (%)*	225 (56.2%)	4.18.2

* For estimation of food-related cases it was assumed that the proportions derived from expert consultation would exclude travel-related cases

4.18.1 Case definition

Clinical description: An acute illness with diarrhoea, fever and abdominal pain. Mesenteric adenitis may occur and complications include arthritis and systemic infection

Laboratory test for diagnosis: Isolation of *Yersinia enterocolitica* or *Y. pseudotuberculosis* from blood or faeces OR detection of circulating antigen by ELISA or agglutination test

Case classification:

Probable A clinically compatible illness that is either a contact of a confirmed case of the same disease, or has had contact with the same common source i.e., is part of an identified common source outbreak

Confirmed A clinically compatible illness that is laboratory confirmed

4.18.2 Yersiniosis cases reported in 2009 by data source

During 2009, 431 notifications (10.0 cases per 100 000 population) of yersiniosis and no resulting deaths were reported in EpiSurv.

The ICD-10 code A04.6 was used to extract yersiniosis hospitalisation data from the MoH NMDS database. Of the 46 hospital admissions (1.1 admissions per 100 000 population) recorded in 2009, 24 were reported with yersiniosis as the primary diagnosis and 22 with yersiniosis as another relevant diagnosis.

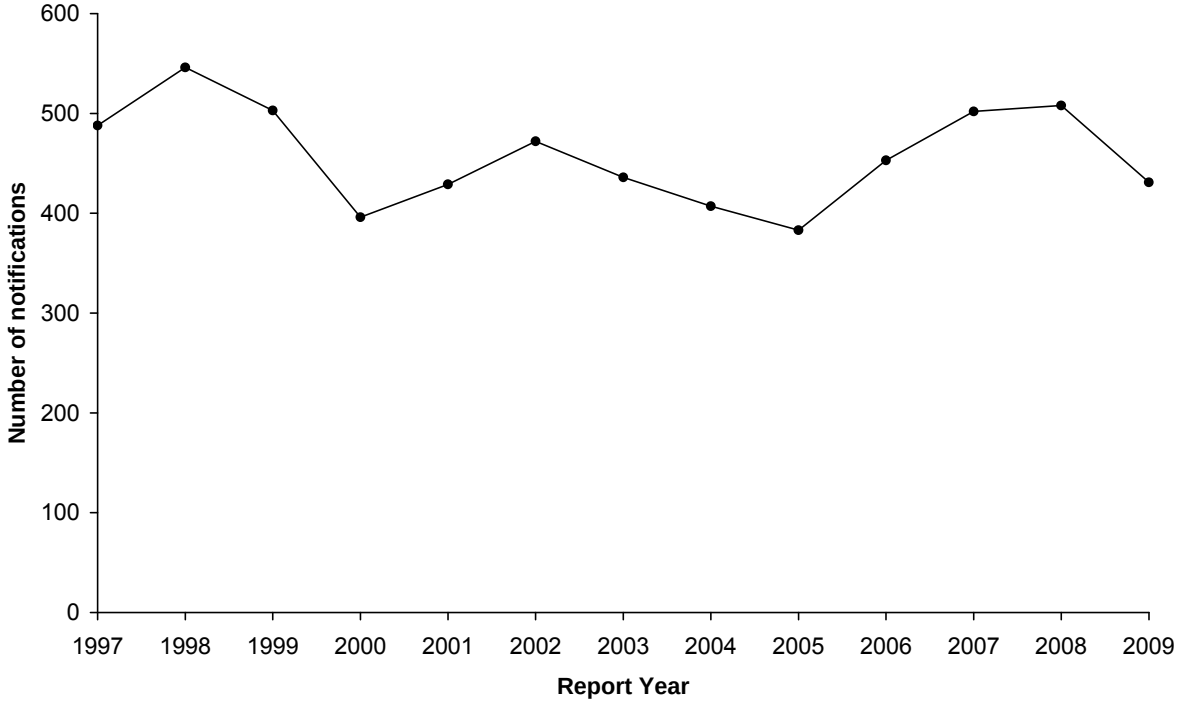
It has been estimated by expert consultation that 56% (minimum = 42%, maximum = 71%) of yersiniosis incidence is due to foodborne transmission. Approximately 50% of foodborne transmission was estimated to be due to consumption of pork.

4.18.3 Notifiable disease data

4.18.3.1 Annual notification trend

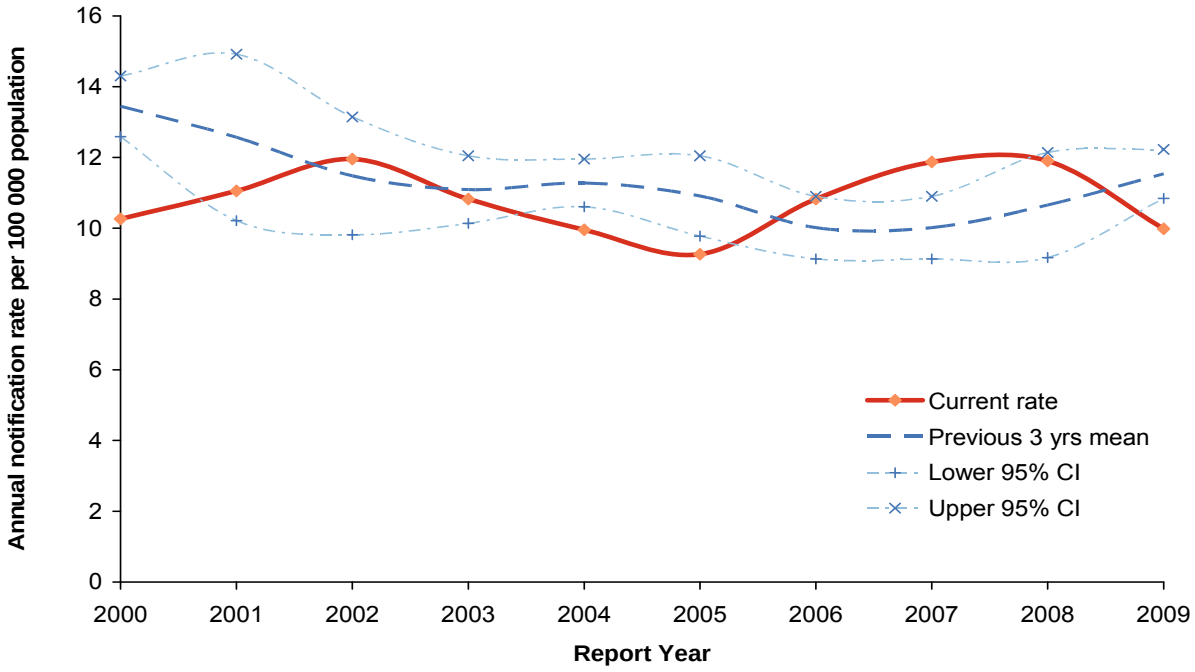
During 2009, 431 yersiniosis notifications were reported in EpiSurv. Yersiniosis became notifiable in 1996, with the highest number of notifications reported in 1998 (546 cases). Since 1997, the annual number of notifications has fluctuated slightly across the years, but has remained between 407 and 546 cases (Figure 55).

Figure 55: Yersiniosis notifications by year, 1997-2009



The yersiniosis notification rate was 10.0 per 100 000 population in 2009. The yersiniosis notification rate has varied little (ranging from 9.3 to 12.0 per 100 000) between 2000 and 2009 (Figure 56).

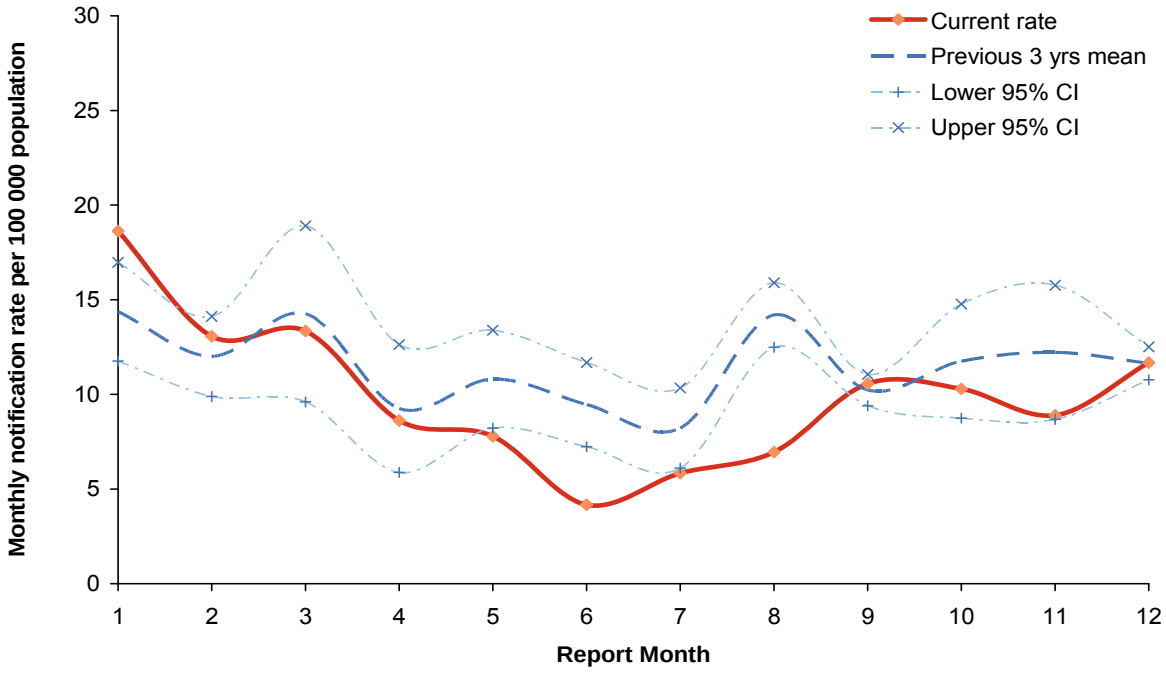
Figure 56: Yersiniosis notification rate by year, 2000-2009



4.18.3.2 Seasonality

The number of notified cases of yersiniosis per 100 000 population by month for 2009 is shown in Figure 57. The 2009 notification rate follows a similar, but more pronounced seasonal pattern in comparison to the historic mean rate, with a summer peak and winter trough.

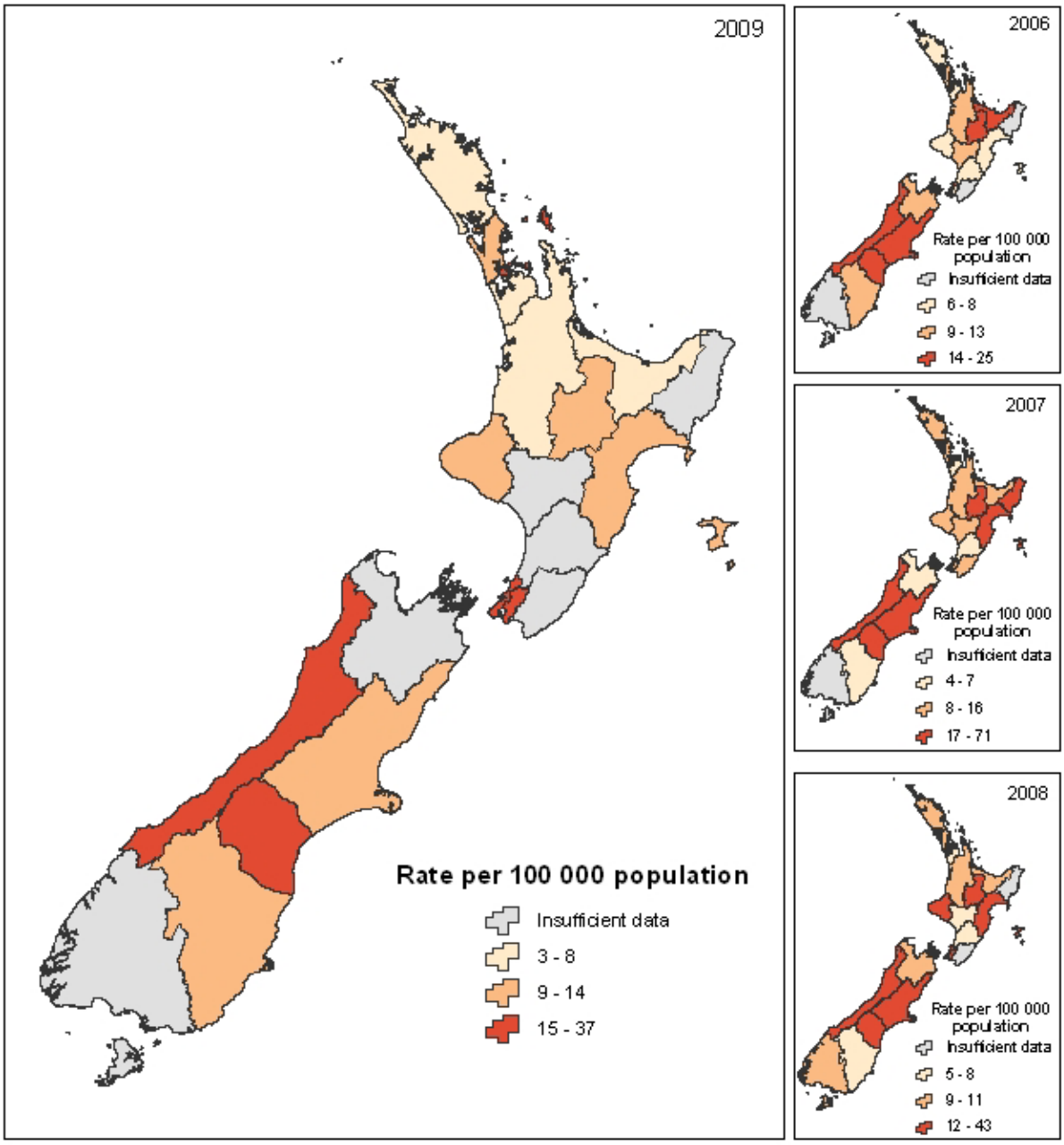
Figure 57: Yersiniosis monthly rate (annualised) for 2009



4.18.3.3 Geographic distribution of yersiniosis notifications

Yersiniosis notification rates vary throughout New Zealand as illustrated in Figure 58. The highest rates were recorded in West Coast (36.8 per 100 000 population, 12 cases), South Canterbury (18.0 per 100 000, 10 cases), and Hutt Valley (17.5 per 100 000, 25 cases) DHBs. West Coast and South Canterbury DHBs have been in the highest quantile of yersiniosis notification rates for each of the last four years.

Figure 58: Geographic distribution of yersiniosis notifications, 2006-2009



4.18.3.4 Age and sex distribution of yersiniosis cases

The yersiniosis notification rate was slightly higher for males than for females in 2009. However, the hospitalisation rate was slightly higher for females (Table 66).

Table 66: Yersiniosis cases by sex, 2009

Sex	EpiSurv notifications		Morbidity[a]		Deaths recorded in EpiSurv
	No.	Rate[b]	No.	Rate[b]	No.
Male	220	10.4	18	0.9	
Female	207	9.4	28	1.3	
Unknown	4				
Total	431	10.0	46	1.1	

[a] MoH morbidity data for hospital admissions
[b] per 100 000 of population

In 2009, the highest age-specific yersiniosis notification rates occurred in the less than one year (65.0 per 100 000 population, 41 cases) and the 1 to 4 years (38.8 per 100 000, 94 cases) age groups. Age-specific notifications rates were more than three times higher for those groups than for any other age group (Table 67). The highest hospitalisation rates were reported for those in the less than one year age group, although hospitalisation rates were not calculated for most age groups, due to the small numbers of cases.

Table 67: Yersiniosis cases by age group, 2009

Age group	EpiSurv notifications		Morbidity[a]		Deaths recorded in EpiSurv
	No.	Rate[b]	No.	Rate[b]	No.
<1	41	65.0	5	7.9	
1 to 4	94	38.8	2	-	
5 to 9	19	6.6	4	-	
10 to 14	14	4.7	0	-	
15 to 19	17	5.3	0	-	
20 to 29	40	6.8	7	1.2	
30 to 39	42	7.3	1	-	
40 to 49	48	7.6	5	0.8	
50 to 59	45	8.5	3	-	
60 to 69	36	9.2	3	-	
70+	34	8.9	16	4.2	
Unknown	1				
Total	431	10.0	46	1.1	

[a] MoH morbidity data for hospital admissions
[b] per 100 000 of population. Where fewer than five cases have been reported a rate has not been calculated.

4.18.3.5 Risk factors reported

In 2009, the most commonly reported risk factors for yersiniosis notifications were consumption of food from retail premises (40.9%) and contact with farm animals (36.6%) (Table 68).

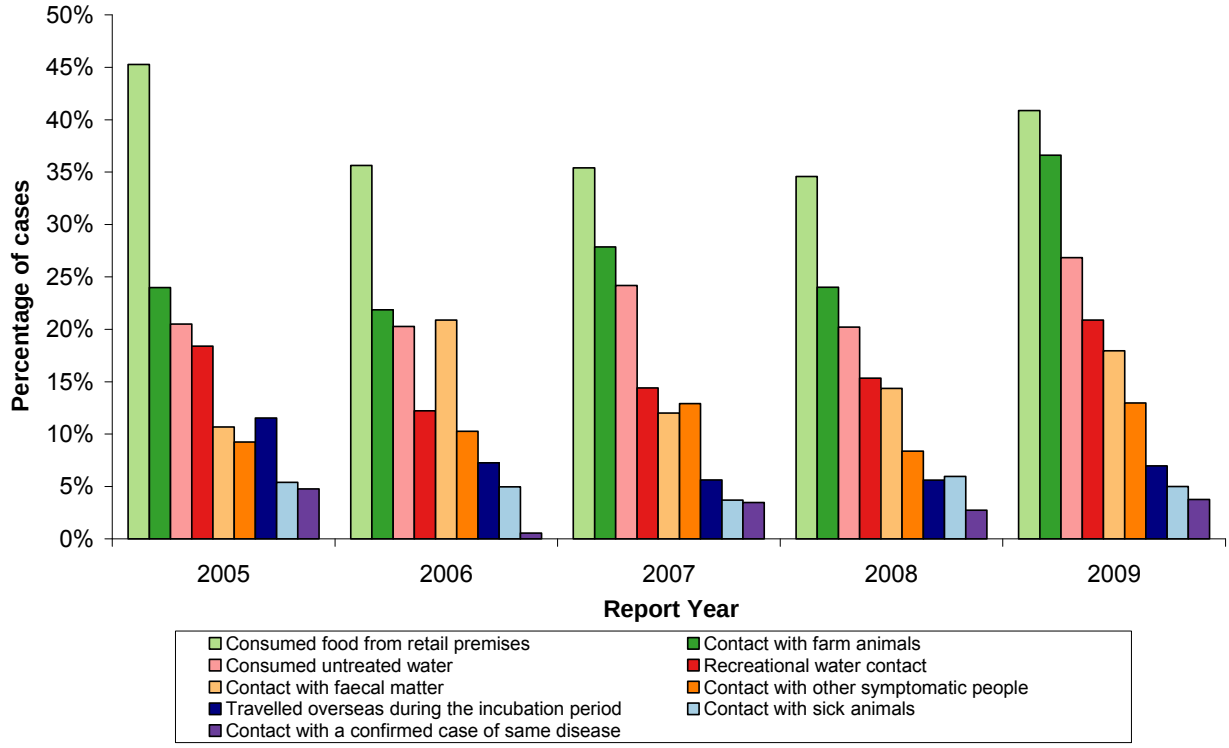
Table 68: Exposure to risk factors associated with yersiniosis, 2009

	Notifications			
Risk Factor	**Yes**	**No**	**Unknown**	**%[a]**
Consumed food from retail premises	47	68	316	40.9
Contact with farm animals	52	90	289	36.6
Consumed untreated water	29	79	323	26.9
Recreational water contact	24	91	316	20.9
Contact with faecal matter	21	96	314	17.9
Contact with other symptomatic people	17	114	300	13.0
Travelled overseas during the incubation period	11	147	273	7.0
Contact with sick animals	6	114	311	5.0

[a]Percentage refers to the cases that answered "yes" out of the total number of cases for which this information was supplied. Cases may have more than one risk factor recorded.

Between 2005 and 2009, the risk factors associated with yersiniosis cases have generally occurred in the same order of importance each year (Figure 59). Over the past five years, consumption of food from retail premises has been the most commonly reported risk factor. The trend suggests a growing importance of contact with farm animals as a risk factor associated with yersiniosis cases.

Figure 59: Yersiniosis risk factors by percentage of cases and year, 2005-2009



4.18.3.6 Estimate of travel-related cases

For cases where information on travel was provided, 7.0% (95%CI 3.5-12.1%) had travelled overseas during the incubation period. Assuming that the cases for which travel information was provided were representative of all yersiniosis cases, a Poisson distribution can be used to estimate the total number of potentially travel related cases of yersiniosis in 2009. The resultant distribution has a mean of 30 cases (95% CI 15-49).

If data from the last four years are considered, the estimated proportion of cases travelling overseas within the incubation period of the organism is 6.4% (95% CI 5.0-8.1%).

4.18.4 Outbreaks reported as caused by *Yersinia* spp.

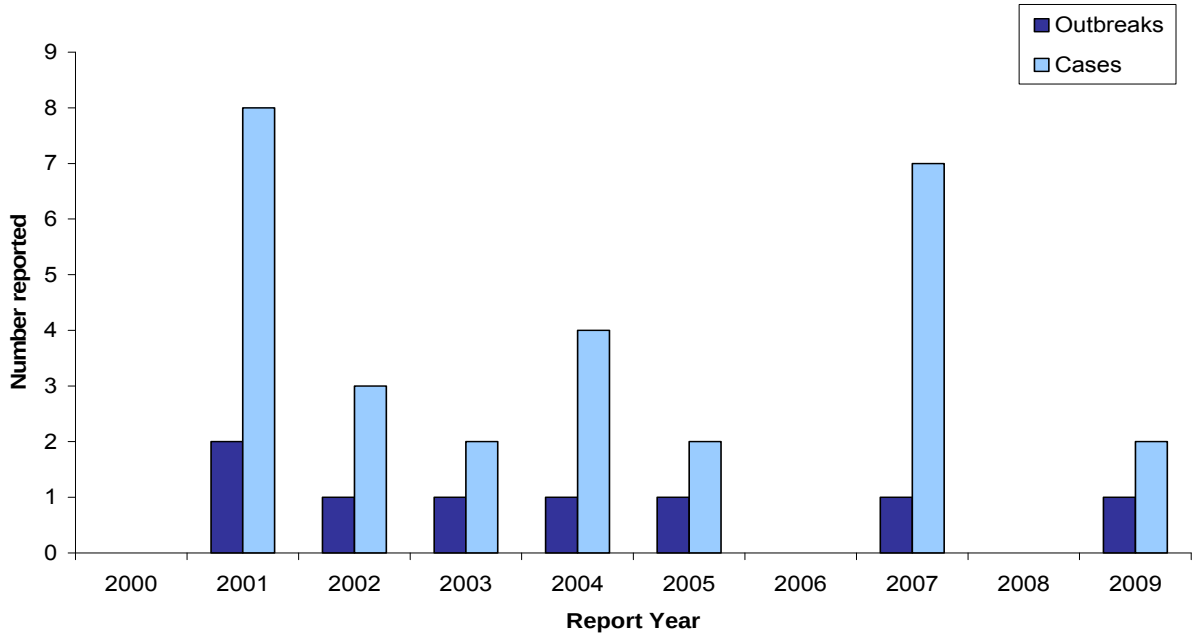
During 2009, there were two *Yersinia* spp. outbreaks reported in EpiSurv, with one of these associated with a suspected foodborne source (Table 69). Two cases were associated with this outbreak.

Table 69: *Yersinia* spp. outbreaks reported, 2009

Measure (No.)	Foodborne *Yersinia* spp. outbreaks	All *Yersinia* spp. outbreaks
Outbreaks	1	2
Cases	2	15
Hospitalised cases	0	0

Between 2000 and 2009 very few foodborne *Yersinia* spp. outbreaks were reported in EpiSurv (two or less each year), with a small total number of associated cases (ranging from two to eight) (Figure 60).

Figure 60: Foodborne *Yersinia* outbreaks and associated cases reported by year, 2000 – 2009



4.18.4.1 Details of food-associated outbreaks

Table 70 contains details of the food–associated *Yersinia* spp. outbreak reported in 2009.

Table 70: Details of the food-associated *Yersinia* spp. outbreak, 2009

Public Health Unit (Month)	Suspected vehicle	Setting	Number ill	Confirmation
West Coast (September)	Pork and chicken	Restaurant/Café	1C, 1P	2

C = confirmed, P = probable
Confirmation:
1 = Environmental investigation – identified critical control point failures linked to implicated source
2 = Epidemiological – case had history of exposure to implicated source
3 = Epidemiological – case control or cohort study showed elevated risk for cases to implicated source
4 = Laboratory – pathogen suspected to have caused illness identified in food handler
5 = Laboratory – pathogen suspected to have caused illness identified in implicated source (food)
6 = No evidence
7 = Other evidence

The *Yersinia* spp. outbreak was linked to the consumption of inadequately prepared pork and chicken. Exposure occurred while the cases were overseas (Samoa). Contact with a wild pig was also reported during the incubation period of the disease.

4.18.4.2 Laboratory investigation of samples from suspected foodborne outbreaks

During investigations of suspected foodborne illness outbreaks by ESR's Public Health Laboratory, no samples were found to contain *Yersinia* spp.

4.18.5 Relevant New Zealand studies and publications

Nil.

4.18.6 Relevant regulatory developments

Nil.

5 SUMMARY TABLES

This appendix brings together data from different sources as summary tables to facilitate comparisons between conditions.

Table 71: Number of cases and rates per 100 000 population of selected notifiable diseases in New Zealand during 2008 and 2009

Disease	2008		2009		
	Cases	Rates	Cases	Rates	Change [b,c]
Campylobacteriosis	6 694	156.8	7 176	166.3	➔
Cryptosporidiosis	764	17.9	854	19.8	➔
Gastroenteritis [a]	687	16.1	714	16.5	→
Giardiasis	1 660	38.9	1 640	38.0	←
Hepatitis A	89	2.1	44	1.0	←
Listeriosis	27	0.6	28	0.6	→
Salmonellosis	1 345	31.5	1 129	26.2	←
Shigellosis	113	2.6	119	2.8	→
VTEC/STEC Infection	124	2.9	143	3.3	→
Yersiniosis	508	11.9	431	10.0	←

[a] Cases of gastroenteritis from a common source or foodborne intoxication e.g. staphylococcal intoxication
[b] ←= Significant decrease, ➔ = Significant increase, ▫ = No change, ← = Not significant decrease, → = not significant increase, NA = not applicable
[c] The Mantel-Haenszel chi-square test or where necessary Fisher's Exact test were used to determine statistical significance. P-values less than 0.05 are considered to be significant at the 95% level of confidence.

Table 72: Deaths due to selected notifiable diseases recorded in EpiSurv, 1997-2009

Disease	1997	1998	1999	2000	2001	2002	2003	2004	2005	2006	2007	2008	2009
Campylobacteriosis	2	2	1	3	1	1	0	0	1	1	1	0	0
Gastroenteritis	0	0	0	0	0	1	0	0	0	0	0	0	0
Giardiasis	1	0	0	0	0	0	0	0	0	0	0	0	0
Listeriosis - non perinatal	2	0	1	2	1	0	2	3	1	0	2	3	2
Listeriosis - perinatal	6	0	2	4	1	3	2	2	0	1	2	2	2
Salmonellosis	2	2	1	7	2	1	0	0	1	1	1	1	1
Shigellosis	0	0	1	0	0	0	0	0	0	0	0	0	0
VTEC/STEC infection	1	1	0	0	0	0	0	0	0	0	0	0	1
Yersiniosis	0	2	0	0	0	0	0	1	0	0	0	0	0

Note: The numbers in this table are those recorded in EpiSurv where the notifiable disease was the primary cause of death. Information on deaths is most likely to be reported by Public Health Services when it occurs close to the time of notification and investigation.

Table 73: MoH mortality data for selected notifiable diseases, 2005-2007

Disease	ICD 10 Codes	2005		2006		2007 [a]	
		Underlying[b]	Contributory[c]	Underlying[b]	Contributory[c]	Underlying[b]	Contributory[c]
Campylobacteriosis	A04.5	0	3	3	0	1	0
Cryptosporidiosis	A072	0	0	0	0	0	0
Giardiasis	A07.1	0	0	0	0	0	0
Hepatitis A	B15	0	0	0	0	0	2
Listeriosis	A32	0	0	0	1	2	0
Salmonellosis	A02	0	1	1	0	0	0
Shigellosis	A03	0	0	0	0	0	0
Yersiniosis	A04.6	0	0	0	0	0	0

[a] Latest year that data are available

[b] Underlying – main cause of death

[c] Contributory – selected contributory cause of death (not main cause of death)

Table 74: MoH morbidity data for selected notifiable diseases, 2007-2009

Disease	ICD 10 Codes	2007		2008		2009	
		Principal diagnosis	Other relevant diagnosis	Principal diagnosis	Other relevant diagnosis	Principal diagnosis	Other relevant diagnosis
Campylobacteriosis	A04.5	752	185	388	97	473	101
Cryptosporidiosis	A07.2	26	14	19	13	19	4
Giardiasis	A07.1	20	14	18	21	21	13
Hepatitis A	B15	17	18	19	18	17	7
Listeriosis	A32	12	17	13	13	11	17
Salmonellosis	A02	123	27	118	40	130	28
Shigellosis	A03	27	1	15	4	14	5
Toxic shellfish poisoning	T61.2	6	1	6	0	0	0
VTEC/STEC infection	A04.3	22	24	26	20	24	11
Yersiniosis	A04.6	19	31	23	30	24	22

Note: Hospital admission data may include multiple admissions (to the same or different hospitals) for the same case and admissions may relate to cases first diagnosed in previous years.

Table 75: Number of cases and rates of selected notifiable diseases per 100 000 population by ethnic group, 2009

	Ethnicity											
	European		Māori		Pacific Peoples		Asian		Other Ethnicity		Unknown	
Disease	Cases	Rate	Cases	Rate	Cases	Rate	Cases	Rate	Cases	Rate	Cases	Rate
Campylobacteriosis	5 389	200.0	461	81.5	172	76.0	398	116.8	39	115.1	717	
Cryptosporidiosis	705	26.2	65	11.5	18	8.0	23	6.7	4		39	
Gastroenteritis	505	18.7	39	6.9	10	4.4	36	10.6	6	17.7	118	
Giardiasis	1 265	47.0	94	16.6	12	5.3	76	22.3	55	162.4	138	
Hepatitis A	18	0.7	6	1.1	1		15	4.4	2		2	
Listeriosis	15	0.6	5	0.9	3		1		4			
Salmonellosis	814	30.2	126	22.3	37	16.4	77	22.6	8	23.6	67	
Shigellosis	56	2.1	5	0.9	28	12.4	18	5.3	2		10	
VTEC/STEC infection	111	4.1	20	3.5	3		2		1		6	
Yersiniosis	247	9.2	37	6.5	12	5.3	98	28.8	2		35	

Note: Disease rates for ethnic groups are based on 2006 census data from Statistics New Zealand and should not be compared to disease rates used elsewhere in the report, which have been calculated using 2009 mid-year population estimates from Statistics New Zealand. Where fewer than five cases have been notified, a rate has not been calculated and the cell has been left blank.

Table 76: Number of cases and rates of selected notifiable diseases per 100 000 population by sex, 2009

	Sex							
	Male		Female		Unknown		Total	
Disease	Cases	Rate	Cases	Rate	Cases	Rate	Cases	Rate
Campylobacteriosis	3 976	187.8	3 119	141.9	81		7 176	166.3
Cryptosporidiosis	384	18.1	463	21.1	7		854	19.8
Gastroenteritis	276	13.0	407	18.5	31		714	16.5
Giardiasis	838	39.6	781	35.5	21		1 640	38.0
Hepatitis A	26	1.2	18	0.8	0		44	1.0
Listeriosis – non perinatal	6	0.3	12	0.5	0		18	0.4
Salmonellosis	555	26.2	564	25.7	10		1 129	26.2
Shigellosis	57	2.7	61	2.8	1		119	2.8
VTEC/STEC infection	59	2.8	82	3.7	2		143	3.3
Yersiniosis	220	10.4	207	9.4	4		431	10.0

Table 77: Number of cases and rates of selected notifiable diseases per 100 000 population by age group, 2009

	Age Group																							
	<1		1 to 4		5 to 9		10 to 14		15 to 19		20 to 29		30 to 39		40 to 49		50 to 59		60 to 69		70+		Unknown		Total	
Disease	Cases	Rate	Cases	Rate	Cases	Rate	Cases	Rate	Cases	Rate	Cases	Rate	Cases	Rate	Cases	Rate	Cases	Rate	Cases	Rate	Cases	Rate	Cases	Rate	Cases	Rate
Campylobacteriosis	156	247.3	818	337.4	370	128.4	325	109.2	495	153.2	1262	215.7	838	145.4	887	139.7	773	145.5	666	169.5	563	147.8	23		7 176	166.0
Cryptosporidiosis	29	46.0	267	110.1	102	35.4	69	23.2	44	13.6	93	15.9	131	22.7	58	9.1	30	5.6	15	3.8	13	3.4	3		854	19.8
Gastroenteritis	31	49.2	87	35.9	7	2.4	12	4.0	18	5.6	67	11.5	64	11.1	87	13.7	80	15.1	53	13.5	171	44.9	37		714	16.5
Giardiasis	36	57.1	331	136.5	127	44.1	55	18.5	23	7.1	153	26.2	373	64.7	243	38.3	153	28.8	111	28.3	26	6.8	9		1 640	38
Hepatitis A			2		4		6	2.0	3		6	1.0	4		9	1.4	6	1.1	4						44	1.0
Listeriosis							1		2		5	0.9	4		1		2		3		10	2.6			28	0.6
Salmonellosis	78	123.7	218	89.9	68	23.6	38	12.8	60	18.6	144	24.6	136	23.6	121	19.1	115	21.6	72	18.3	75	19.7	4		1 129	26.2
Shigellosis	2		11	4.5	8	2.8	4		4		25	4.3	17	2.9	18	2.8	12	2.3	12	3.1	6	1.6			119	2.8
VTEC/STEC infection	9	14.3	53	21.9	15	5.2	3		9	2.8	12	2.1	8	1.4	7	1.1	11	2.1	9	2.3	7	1.8			143	3.3
Yersiniosis	41	65.0	94	38.8	19	6.6	14	4.7	17	5.3	40	6.8	42	7.3	48	7.6	45	8.5	36	9.2	34	8.9	1		431	10.0

Note: Where fewer than five cases have been notified a rate has not been calculated and the cell has been left blank.

Rates for each disease have been divided into three quantiles (tertiles) and shaded to indicate the age groups with highest, medium and lowest rates of disease. Shadings used are:

- Fewer than 5 cases
- First (lowest) tertile
- Second (middle) tertile
- Third (highest) tertile

Table 78: Number of cases and rates of selected notifiable diseases per 100 000 population by District Health Board, 2009

Notifications

District Health Board	Northland	Waitemata	Auckland	Counties Manukau	Waikato	Lakes	Bay of Plenty	Tairawhiti	Taranaki	Hawke's Bay	Whanganui	MidCentral	Hutt	Capital and Coast	Wairarapa	Nelson Marlborough	West Coast	Canterbury	South Canterbury	Otago	Southland	Total
Campylobacteriosis	208	913	810	685	661	205	317	36	233	336	139	236	355	692	69	173	42	545	103	253	165	7176
Cryptosporidiosis	37	74	56	51	109	13	27	10	24	36	9	29	19	129	13	24	18	97	28	28	23	854
Gastroenteritis	3	82	98	44	24	12	5			11	16	108	53	89	2	10	90	52	3	9	3	714
Giardiasis	64	155	212	202	148	65	63	11	45	66	21	18	35	167	12	48	13	190	10	51	44	1640
Hepatitis A	1	3	9	6	3	1	2			4		2	3	3				6			1	44
Listeriosis		4	4	2	4		1	2	1		1	2	1	1				2		3		28
Salmonellosis	32	88	106	100	104	27	41	32	20	58	12	31	35	59	19	40	10	149	34	76	56	1129
Shigellosis	3	23	19	26	7		4	1		1		1	4	8	1	2	1	10	1	4	3	119
VTEC/STEC infection	6	9	18	9	27	5	9	4	14	4		3	2	5	1	2	1	16	2	4	2	143
Yersiniosis	11	51	69	34	28	10	6	3	15	15	3	4	25	42	1	2	12	67	10	19	4	431

Rates

District Health Board	Northland	Waitemata	Auckland	Counties Manukau	Waikato	Lakes	Bay of Plenty	Tairawhiti	Taranaki	Hawke's Bay	Whanganui	MidCentral	Hutt	Capital and Coast	Wairarapa	Nelson Marlborough	West Coast	Canterbury	South Canterbury	Otago	Southland	Total
Campylobacteriosis	133.5	172.8	182.4	142.2	183.6	201.4	152.6	77.9	215.3	218.3	220.1	142.3	248.8	240.2	172.6	126.5	128.9	108.6	185.4	134.2	147.6	166.3
Cryptosporidiosis	23.8	14.0	12.6	10.6	30.3	12.8	13.0	21.6	22.2	23.4	14.3	17.5	13.3	44.8	32.5	17.5	55.2	19.3	50.4	14.9	20.6	19.8
Gastroenteritis		15.5	22.1	9.1	6.7	11.8	2.4			7.1	25.3	65.1	37.1	30.9		7.3	276.2	10.4		4.8		16.5
Giardiasis	41.1	29.3	47.7	41.9	41.1	63.9	30.3	23.8	41.6	42.9	33.3	10.8	24.5	58.0	30.0	35.1	39.9	37.9	18.0	27.1	39.3	38.0
Hepatitis A			2.0	1.2														1.2				1.0
Listeriosis																						0.6
Salmonellosis	20.5	16.7	23.9	20.8	28.9	26.5	19.7	69.3	18.5	37.7	19.0	18.7	24.5	20.5	47.5	29.2	30.7	29.7	61.2	40.3	50.1	26.2
Shigellosis		4.4	4.3	5.4	1.9									2.8				2.0				2.8
VTEC/STEC infection	3.9	1.7	4.1	1.9	7.5	4.9	4.3		12.9					1.7				3.2				3.3
Yersiniosis	7.1	9.7	15.5	7.1	7.8	9.8	2.9		13.9	9.7			17.5	14.6			36.8	13.3	18.0	10.1		10.0

Rates for each disease have been divided into three quantiles (tertiles) and shaded to indicate DHBs with the highest, middle and lowest rates of disease. Shadings used are:

- Fewer than 5 cases
- First (lowest) tertile
- Second (middle) tertile
- Third (highest) tertile

Table 79: Notifiable disease cases by year, 1987-2009

Note: cell is blank where data are unavailable

Disease	1987	1988	1989	1990	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000	2001	2002	2003	2004	2005	2006	2007	2008	2009
Campylobacteriosis	2 921	2 796	4 187	3 850	4 148	5 144	8 101	7 714	7 442	7 635	8 924	11 572	8 161	8 418	10 146	12 494	14 788	12 215	13 836	15 873	12 778	6 694	7 176
Cryptosporidiosis										119	357	866	977	775	1 208	975	817	611	889	737	924	764	854
Gastroenteritis										555	310	492	601	727	940	1 087	1 026	1 363	557	937	622	687	714
Giardiasis										1 235	2 127	2 183	1 793	1 688	1 604	1 547	1 570	1 514	1 231	1 214	1 402	1 660	1 640
Hepatitis A	158	176	134	150	224	288	257	179	338	311	347	145	119	107	61	106	70	49	51	123	42	89	44
Listeriosis	12	7	10	16	26	16	11	8	13	10	35	17	19	22	18	19	24	26	20	19	26	27	28
Salmonellosis	1 140	1 128	1 860	1 619	1 244	1 239	1 340	1 522	1 334	1 141	1 177	2 069	2 077	1 795	2 417	1 880	1 401	1 081	1 382	1 335	1 275	1 345	1 129
Shigellosis	143	145	137	197	152	124	128	185	191	167	117	122	147	115	157	112	87	140	183	102	129	113	119
VTEC/STEC infection							3	3	6	7	13	48	64	67	76	73	104	89	92	87	100	124	143
Yersiniosis										330	488	546	503	396	429	472	436	407	383	453	502	508	431

Table 80: Rates per 100 000 population of selected notifiable diseases in New Zealand and other selected countries

Disease	New Zealand	Australia[1]	USA[2]	Canada[4]	UK[5]	EU Total[5]	Other High
Year	**2009**	**2009**	**2009**	**2006**	**2007**	**2007**	
Campylobacteriosis	166.3	72.5	13.0	29.7 (2005)	95	46.6	235 (Czech Republic)[5] 104.1 (Switzerland)[6]
Cryptosporidiosis	19.8	21.0	2.9	2.2	6.0	2.4	14 (Ireland)[5]
Giardiasis	38.0	NN	7.7[3]	12.2	5.4	62.7	734 (Romania)[5] 71.3 (Tajikistan)[6] 69.4 (Russian Federation)[6]
Hepatitis A	1.0	2.6	1.0[3]	NN	0.6	2.8	229.4 (Kyrgyzstan)[6] 124.3 (Latvia)[6]
Listeriosis	0.6	0.4	0.3	NN	0.4	0.3	3.5 (San Marino)[6] 1.3 (Iceland)[5] 1.1 (Norway)[5]
Salmonellosis	26.2	43.6	15.2	18.0	22	34.3	172 (Czech Republic)[5] 155 (Slovakia)[5]
Shigellosis	2.8	2.8	4.0	2.0	2.9	2.1	53.2 (Kyrgyzstan)[6] 24.3 (Tajikistan)[6]
VTEC/STEC Infection	3.3	0.7	1.6[7]	3.1	1.9	0.6	10.4 (Azerbaijan)[6] 5.5 (Israel)[6] 4.3 (Iceland)[5]
Yersiniosis	10.0	NN	0.3	1.8	0.1	2.9	17 (Lithuania)[5] 9.1 (Finland)[5]

NN Not notifiable

1 National Notifiable Diseases Surveillance System (NNDSS) http://www9.health.gov.au/cda/source/CDA-index.cfm

2 FoodNet – Foodborne Diseases Active Surveillance Network http://www.cdc.gov/foodnet/

3 Centers for Disease Control and Prevention. Summary of notifiable disease http://www.cdc.gov/mmwr/mmwr_nd/index.html (CDC data presented here relate to the 2007 year)

4 National Enteric Surveillance Program (NESP) http://www.nml-lnm.gc.ca/NESP-PNSME/index-eng.htm

5 European Centre for Disease Prevention and Control (ECDC). Annual epidemiological report on communicable diseases in Europe http://ecdc.europa.eu/en/Pages/home.aspx (ECDC data presented here relate to the 2007 year)

6 World Health Organization Regional Office for Europe Centralized Information System for Infectious Diseases (CISID) http://data.euro.who.int/cisid/?TabID=67 (CISID data presented here relates to the 2008 year)

7 Sum of O157 and non-O157

Table 81: Foodborne outbreaks and associated cases by agent type, 2009

Agent type	No. of outbreaks	% of outbreaks (n=85)	No. of cases	% of cases (n=653)
Norovirus	29	34.1	349	53.4
Campylobacter spp.	7	8.2	39	6.0
Salmonella spp.	6	7.1	47	7.2
Clostridium spp.	3	3.5	88	13.5
Vibrio parahaemolyticus	2	2.4	7	1.1
Ciguatera fish poisoning	1	1.2	6	0.9
Histamine (scombroid) fish poisoning	1	1.2	3	0.5
Listeria spp.	1	1.2	2	0.3
Salmonella Paratyphi B	1	1.2	2	0.3
Yersinia spp.	1	1.2	2	0.3
Unidentified pathogen[1]	33	38.8	108	16.5
Total	**85**	**100**	**653**	**100**

[1] All outbreaks with no pathogen identified were classified as gastroenteritis

Table 82: Outbreaks associated with commercial food operators, 2009

Outbreak setting	No. of outbreaks[1]	% of total outbreaks (n=639)	No. of cases[1]	% of total cases (n=10 736)
Restaurant/cafe	69	10.8	286	2.6
Takeaway	16	2.5	47	0.4
Other Food outlet	3	0.5	34	0.3
Supermarket/deli	2	0.3	19	0.2
Caterers	1	0.2	17	0.2

[1] More than one setting was recorded for 138 outbreaks with 3 268 associated cases

Table 83: Foodborne outbreaks and associated cases by implicated food source, 2009

Implicated vehicle / source	No. of outbreaks[1]	% of outbreaks (n=85)	No. of cases	% of cases (n=653)
Poultry	15	17.6	123	18.8
Vegetables (root)	13	15.3	67	10.3
Shellfish[2]	11	12.9	52	8.0
Grains/beans	11	12.9	74	11.3
Fish	11	12.9	53	8.1
Dairy	9	10.6	59	9.0
Rice	6	7.1	104	15.9
Meat (pork)	6	7.1	29	4.4
Vegetables (leafy)	5	5.9	48	7.4
Meat (beef)	4	4.7	21	3.2
Fruits/nuts	4	4.7	24	3.7
Oils/sugars	3	3.5	24	3.7
Vegetables (vine/stalk)	3	3.5	41	6.3
Eggs	2	2.4	29	4.4
Meat (game)	1	1.2	27	4.1
Meat (lamb)	1	1.2	3	0.5
Water	1	1.2	2	0.3
Unspecified food source[3]	18	21.2	153	23.4
No vehicle / source identified	17	20.0	129	19.8

[1] More than one vehicle / source was implicated in some outbreaks
[2] Nine of the 11 shellfish outbreaks were due to oysters only
[3] A common meal, premises or setting may have been implicated but no specific food items were recorded

Note: Mixed foods were assigned to multiple categories based on the groupings published by Painter *et al.* (2009). Only explicit ingredients were assigned into a category. All foods within a mixed item were given equal priority.

Table 84: Foodborne outbreaks by causal agent and implicated vehicle / source, 2009

Implicated vehicle / source[1]	Norovirus	*Campylobacter* spp.	*Salmonella* spp.	*Clostridium* spp.	*Vibrio parahaemolyticus*	Other[2]	Unidenified Pathogen[3]	Total number of outbreaks
Poultry	3	4	0	1	0	1	6	15
Vegetables (root)	6	1	0	1	0	0	5	13
Shellfish	7	0	0	0	1	0	3	11
Grains/beans	4	0	0	0	0	0	7	11
Fish	4	0	0	1	1	3	2	11
Dairy	3	2	0	0	0	0	4	9
Rice	1	0	0	1	0	0	4	6
Meat (pork)	2	1	0	0	0	1	2	6
Vegetables (leafy)	2	0	0	0	0	0	3	5
Meat (beef)	2	2	0	0	0	0	0	4
Fruits/nuts	0	0	1	0	2	0	1	4
Oils/sugars	1	0	0	0	0	0	2	3
Vegetables (vine/stalk)	2	0	0	0	0	0	1	3
Eggs	1	0	0	0	0	0	1	2
Meat (game)	1	0	0	0	0	0	0	1
Meat (lamb)	0	1	0	0	0	0	0	1
Water	0	0	0	0	0	1	0	1
Unspecified food source[4]	6	0	4	0	0	0	8	18
No vehicle / source identified	4	0	1	1	0	0	11	17
Total	**29**	**7**	**6**	**3**	**2**	**5**	**33**	**85**

1 More than one vehicle / source was implicated in some outbreaks
2 Includes all causal agents listed in Table 81 that were implicated in less than two foodborne outbreaks
3 All outbreaks with no pathogen identified in 2009 were classified as gastroenteritis
4 A common meal, premises or setting may have been implicated but no specific food items were recorded

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